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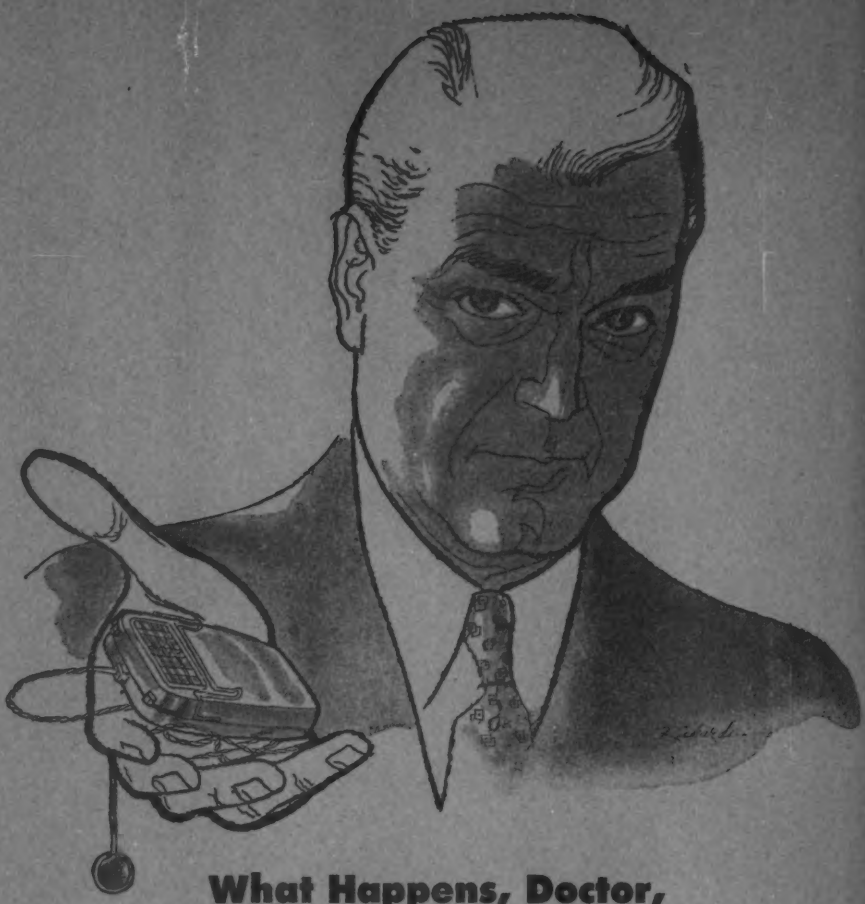

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THE
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VOL. LXIV APRIL, 1954. NO. 4

PRESERVATION OF HEARING IN SURGERY FOR CHRONIC EAR DISEASE.*†

A Consideration of Factors Involved.

ARTHUR L. JUERS, M.D.,
Louisville, Ky.

Several excellent articles have appeared in the literature recently concerning the modified radical mastoidectomy (Bondy operation, atticomastoidectomy). The historical aspects of the subject have been reviewed in detail by previous authors[1,2] and will not be considered further here. This discussion will be concerned chiefly with the factors which influence the preservation or improvement of hearing in this type of ear surgery. In order to discuss the problem adequately, a brief review of the genesis of the lesions amenable to this type of surgery will be necessary.[3]

In general, the case suitable for modified radical mastoidectomy has attic cholesteatoma (cholesteatosis) originating from an attic retraction type of perforation without extension into the middle ear. In some instances the cholesteatoma will eventually spread into the mastoid antrum and mastoid cells in the presence of pneumatization; however, if cholesteatoma has invaded the middle ear to the extent that it cannot be completely exteriorized and yet retain an inflatable tympanic cavity, then a complete radical mastoidectomy is indicated.

A clinical classification of cholesteatoma has been presented previously, together with the mode of genesis of the attic retraction type of cholesteatoma.[3] This phase of the cholesteatoma problem will not be discussed again here, but must be fully understood in order that the technical aspects of surgery in these cases may be fully comprehended.

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* Presented at the meeting of the Cincinnati Society of Otolaryngology, November, 1953.

† From the Department of Otolaryngology, University of Louisville.

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The indications for surgery in attic retraction pathology with suspected cholesteatoma will be briefly reviewed. I believe that there is a trend toward earlier surgery in these cases, because in some instances conservative management results in eventual invasion of the labyrinth and the facial canal; furthermore, the current surgical approach permits the retention of serviceable hearing in a high percentage of cases. Most of the complete radical mastoidectomy operations I have performed in recent years have been on cases of long-standing and, in some instances, neglected attic retraction cholesteatomas in which the tympanic cavity had become filled with cholesteatoma extension.

INDICATIONS FOR SURGERY.

1. Small Attic Opening. Reference to Fig. 1 will reveal the danger of the presence of an attic cholesteatoma with a small attic opening. The exfoliated epithelium from the matrix cannot be extruded through the "bottleneck" at the perforation, and expansion of the lesion internally is inevitable. Conservative treatment of such a lesion will at best merely delay the time when a complication will develop; however, if there has been considerable erosion or absorption of the lateral margin of the opening (floor of the attic and antrum), then conservative management may be justifiably considered. If after a short period of treatment the ear does not become dry, then there undoubtedly is extension of disease beyond the reach of local treatment and surgery is indicated.

2. Persistence of Drainage. This has already been mentioned but deserves being listed as a separate indication. In the younger age groups the persistence of drainage is particularly significant as an indication for surgery. Early surgery in such cases gives the best assurance of retaining useful hearing.

3. Extension into Contiguous Structures. The development of facial, labyrinthine or intracranial extension is an unquestioned indication for immediate surgical intervention.

4. X-ray Evidence of Cholesteatoma. Most cases of attic cholesteatoma treated surgically have not revealed preoperatively the characteristic picture of cholesteatomatous erosion. The conventional X-ray views usually show either sclerosis or, if the mastoid is pneumatized, a cloudiness of the cells.

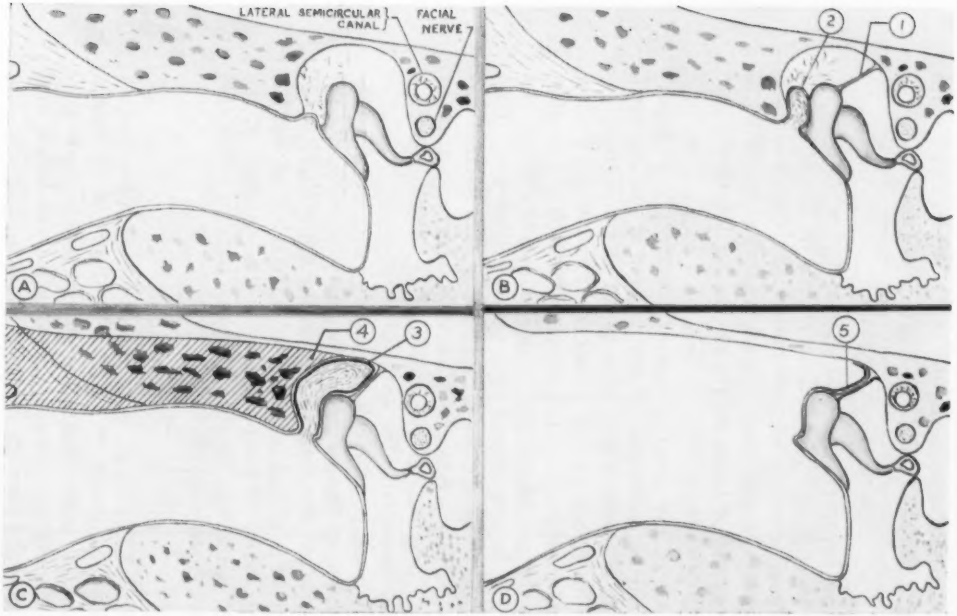


Fig. 1.

A, normal middle ear and external meatus.

B, 1. Connective tissue membrane sealing off the attic from the tympanic cavity. The exact position of this membrane varies considerably. 2. Beginning retraction of Shrapnel's membrane into the attic as a result of negative pressure in the attic. Desquamated epithelium is collecting in the invaginated sac.

C, 3. Cholesteatoma has filled the attic as far as the membrane (B, 1). Anterior and posterior to the plane represented in this diagram the cholesteatoma usually lies against the bony labyrinth and not infrequently against the facial nerve as well. In long-standing cases the cholesteatoma may invade the tympanic cavity. 4. The shaded area is the lateral attic-antral bone which is removed in the modified radical mastoidectomy.

D, cholesteatoma and lateral attic-antral bone has been removed. 5. The medial matrix remains. If this matrix is entirely removed the tympanic cavity must be sealed off with a meatal plastic flap or skin graft.

A minority of cases with proven cholesteatoma show a circumscribed area of erosion. When such an area of erosion is noted on routine X-ray views, the lesion is large and, with few exceptions, the case should be treated surgically.

SURGICAL TECHNIQUE.

I prefer the Lempert endaural approach for surgery in cases where the pathology is located chiefly in the attic and antrum. If there is extensive pneumatization, then the postauricular approach might have some advantage. Good illumination and the use of loupe magnification for the final phase of the operation are essential.

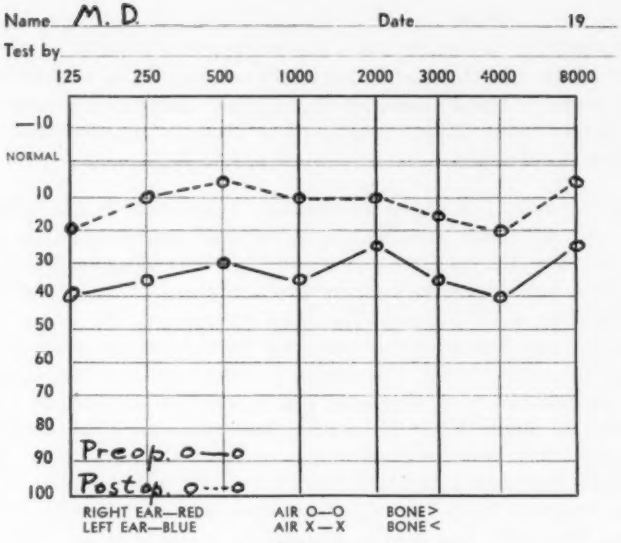
The question of the preservation of the matrix is important in considering technique. If the mastoid is pneumatized, I think that it is preferable to remove all of the matrix, because of the likelihood of the presence of fingerlike projections into underlying cells. In sclerosed mastoids I remove all matrix in the periantral and posterior antral area. I elevate the matrix in the medial attic-antral region and, if no finger-like projections are noted, I preserve this portion unless there is surface evidence of ossicular necrosis such as granulations. Complete exteriorization of the antrum and attic is essential—especially the anterior attic area.

I find it increasingly difficult to justify more or less routine preservation of the matrix. The fact that a 20-decibel hearing level after surgery is frequently maintained, even though the incus is absent, causes one to consider more critically the advisability of compromising on the question of the removal of all pathology, because of uncertainty of being able to retain serviceable hearing.

CLINICAL OBSERVATIONS.

The problem of the integrity of the ossicular chain and its relation to the postoperative hearing level can be discussed best in connection with illustrative case reports. Four possibilities concerning the state of the ossicles are listed, and one or two cases are cited as examples of each situation. The audiometric tests on the operated ear only are shown here.

1. Ossicular Chain Intact. Close inspection of the ossicular chain at surgery revealed that the chain was functionally intact in relatively few. Fig. 2 shows the preoperative and post-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Test by
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NORMAL
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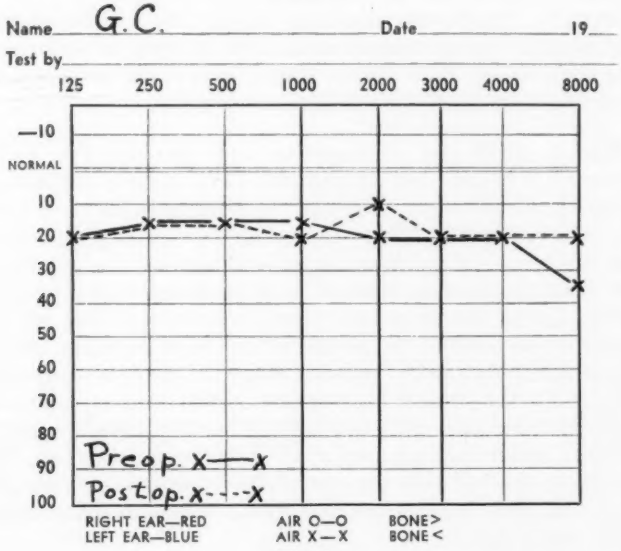
60
70
80
90
100
Preop. o—o
Postop. o---o
RIGHT EAR—RED
LEFT EAR—BLUE
AIR O—O
AIR X—X
BONE>
BONE<

Fig. 2.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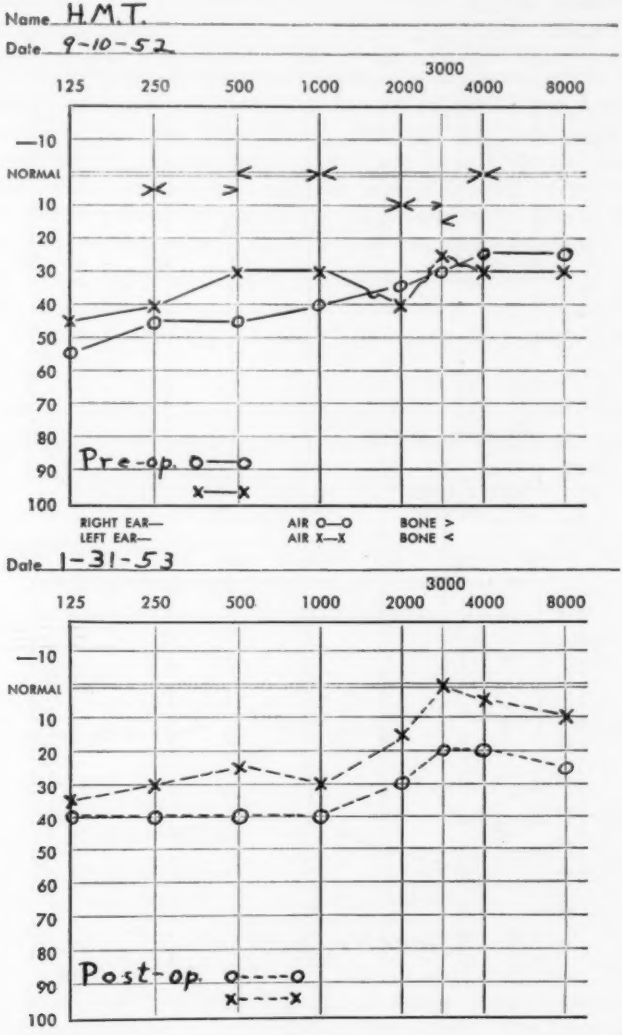
Preop. x—x
Postop. x---x
RIGHT EAR—RED
LEFT EAR—BLUE
AIR O—O
AIR X—X
BONE>
BONE<

Fig. 3.

operative audiogram of a case in which the ossicular chain appeared to be intact under the matrix; the ossicles were not disturbed and the matrix was left *in situ*. This patient was 10 years of age at the time of surgery. There is a possibility that movement of the ossicles may become impeded later by adhesive changes.

2. Ossicles Destroyed by Disease. I find that in the majority of my cases the continuity of the ossicular chain has been disrupted by attic pathology; however, despite this fact, a serviceable level of hearing is maintained in many. Fig. 3 illustrates such a case. The long process of the incus was absent, and the posterior superior portion of the pars tensa was in contact with the upper promontory and the stapes. Cholesteatoma was extensive, and finger-like projections were found in some cells. All matrix was removed. The remains of the incus and the head of the malleus were removed. The middle ear was sealed off from the attic with a small skin flap rotated from the anterior superior meatal wall. This case illustrates the degree of hearing possible when there is contact between the pars tensa and the stapes, in the absence of a functioning ossicular chain. The tympano-stapedial contact was not disturbed and the hearing after surgery was the same as that before. Inflation revealed an inflatable air-containing space over the round window.

Fig. 4 shows the preoperative and postoperative audiograms on a 20-year-old patient who had a history of bilateral otorrhea since two years of age. In both ears the pars tensa was adherent to the upper three-fourths of the promontory, and the stapes was observed as pushing out the overlying pars tensa in tent-pole fashion. Both ears were operated upon, and the incus was completely gone in each ear. The marked degree of adhesion of the pars tensa to the promontory around the stapes undoubtedly impeded stapedial movement and accounted for the greater hearing loss in this case, as compared to the previous one. Had this patient been operated on earlier in life, a better hearing level might have been preserved. The matrix on the left side was completely removed, because of a projection deep into the petrous pyramid behind the superior semicircular canal.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3000
4000
8000
—10
NORMAL
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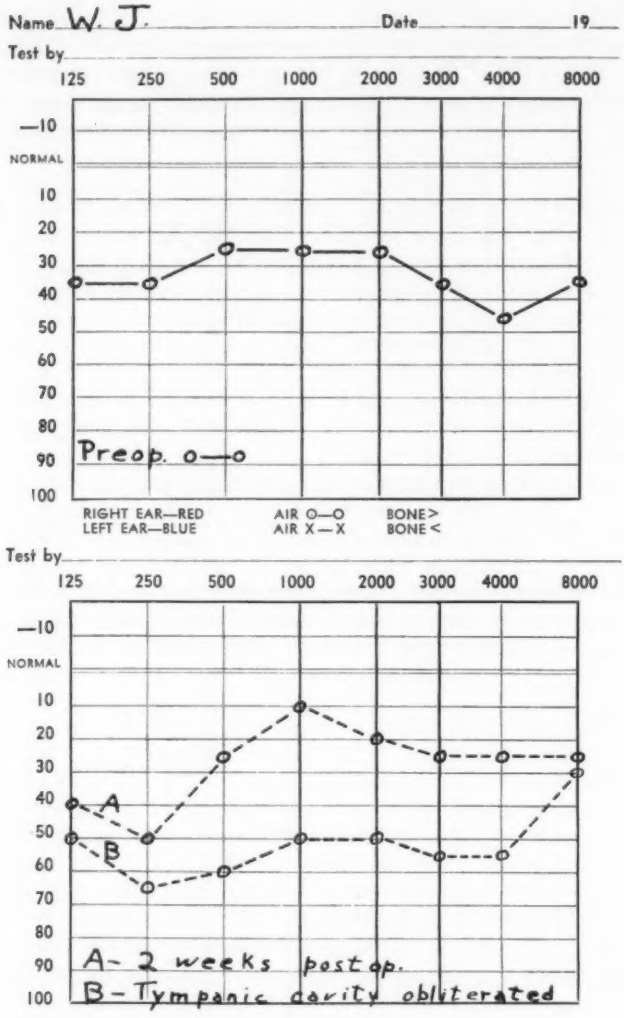
30
40
50
60
70
80
90
100
Pre-op. o—o
x—x
RIGHT EAR—
LEFT EAR—
AIR O—O
AIR X—X
BONE >
BONE <
Date 1-31-53
Post-op. o----o
x----x

Fig. 4.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Preop. o—o
RIGHT EAR—RED
LEFT EAR—BLUE
AIR O—O
AIR X—X
BONE>
BONE<
Test by
125 250 500 1000 2000 3000 4000 8000
—10
NORMAL
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20
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40
50
60
70
80
90
100
A
B
A- 2 weeks post op.
B- Tympanic cavity obliterated

Fig. 5.

Fig. 5 shows the audiograms on another patient in whom the long process of the incus was partially destroyed by pathology. After surgery a serviceable level of hearing was preserved; however, a few months later the tympanic cavity became obliterated as a result of the entire pars tensa becoming adherent to the promontory. Inflation could no longer be accomplished, and the hearing dropped to a 50-decibel level. This illustrates that an inflatable air-containing space over the round window is one of the prerequisites to having serviceable hearing after a modified radical mastoidectomy.

3. Ossicular Chain Fixed—Removed at Operation. In one case, the preoperative audiogram indicated a complete conduction deafness (see Fig. 6). The Eustachian tube inflated, and

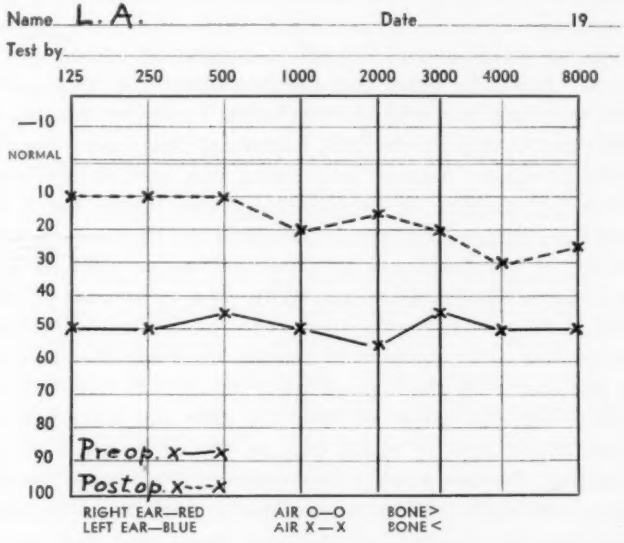
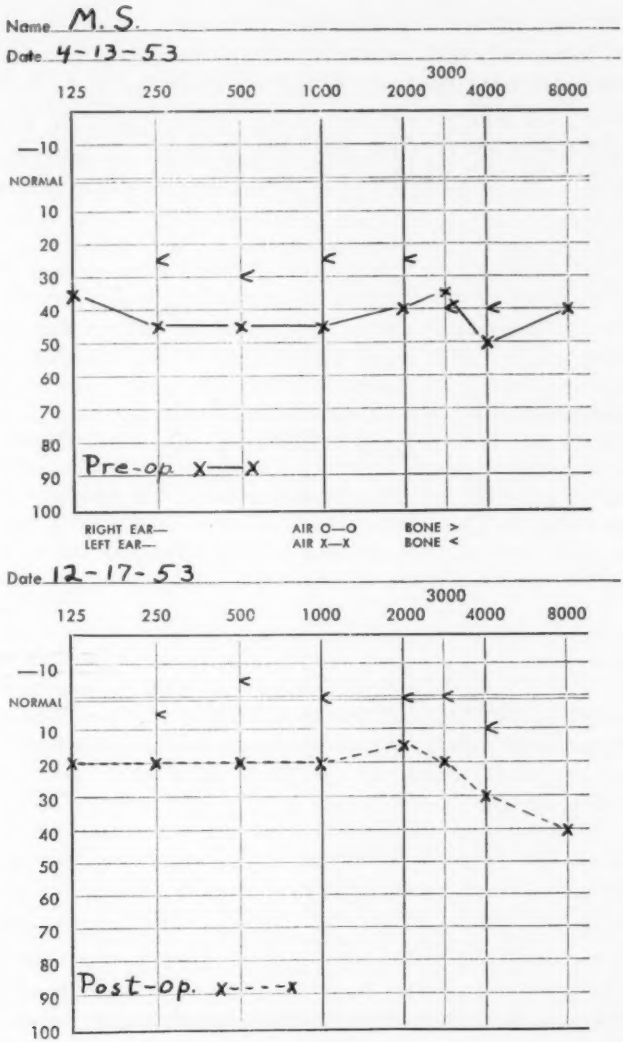


Fig. 6.

the middle ear was free of fluid. At surgery the incudo-malleolar articulation was found to be ankylosed. The incus and head of the malleus were removed. The hearing after surgery came up to a 20-decibel level.

4. Ossicular Chain Intact and Functioning—Removal of Incus Necessary to Permit Removal of Pathology. In the past there has at times been a tendency to compromise with complete removal of pathology, for fear of disturbing a well functioning ossicular chain. Conversely, there has been a tendency by others under these circumstances to do a complete radical mastoidectomy, under the erroneous conception that the removal of the incus would produce a 50-decibel loss anyway. Since it is possible to have 20-decibel hearing level without the presence of the incus, neither the compromise nor the unnecessarily radical surgery is justified.

Fig. 7 shows the audiograms on a patient with a recurrent mastoiditis. A simple mastoidectomy had been done two years previously. Following swimming there had been a recurrence of otorrhea and symptoms suggesting serous labyrinthitis. It will be noted that before surgery the bone conduction threshold was somewhat elevated. Preoperatively the air-bone gap is only 20 decibels, indicating fairly good ossicular chain function. A small perforation was present in the pars tensa. At surgery no cholesteatoma was found, but a pyogenic membrane lined the antrum and the previous operative cavity. It was decided to do a modified radical mastoidectomy in order to eliminate any possibility of a recurrence of infection in the antrum in the event that an otitis media recurred. There was some question of osteitis involving the incus and it, together with the head of the malleus, was removed. The middle ear was sealed off from the attic and antrum with a fenestration type of metal flap, as there was no attic perforation. Postoperatively, the hearing did not reach the optimum level until the central perforation healed and the posterior superior quadrant of the pars tensa retracted and apparently established contact with the stapes. It will be noted that the bone conduction acuity improved after the elimination of the infection. The patient stated that the postoperative hearing level was equal to that present before the development of the otitis media.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—10
NORMAL
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Pre-op. x—x
RIGHT EAR—
LEFT EAR—
AIR O—O
AIR X—X
BONE >
BONE <
Date 12-17-53
125 250 500 1000 3000 2000 4000 8000
—10
NORMAL
10 20 30 40 50 60 70 80 90 100
Post-op. x----x

Fig. 7.

In Fig. 8 are shown the audiograms of a patient in whom the incus and malleus head had to be sacrificed in order to remove cholesteatoma which had extended medial to the ossicles. All matrix was removed. The postoperative audiogram shows an air-bone difference of about 30 decibels instead of the usual 20 noted in most cases in which the incus

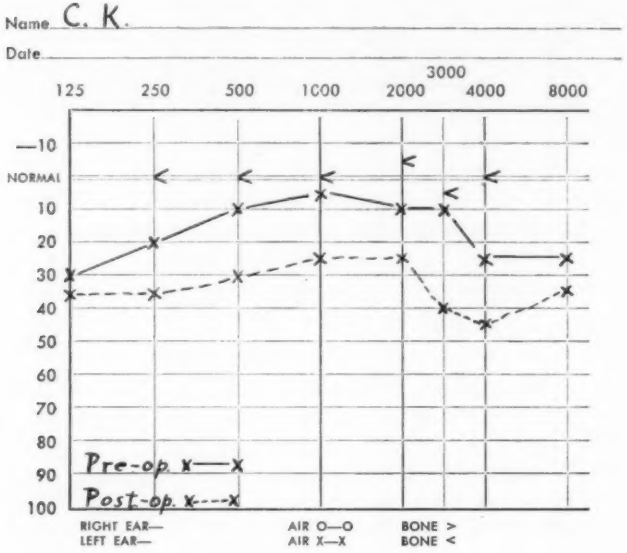

Fig. 8.

was absent. It is my belief that in this case there may have been poor contact between the pars tensa and the stapes after healing took place. In view of this possibility I am suggesting a surgical procedure which I plan to carry out in similar situations in the future to insure contact between the pars tensa and the stapes. Such contact should assure an excellent chance of obtaining a 20-decibel postoperative level if the bone conduction threshold is normal and the mobility of the stapes is unimpeded.

MYRINGOSTAPEDIOPEXY.*

This procedure is suggested when the following situations exist after the pathology has been removed in performing a modified radical mastoidectomy:

1. A functioning incus has been removed for pathological reasons and the pars tensa is not in contact with the stapes.

2. A non-functioning (ankylosed) incus has been removed.

The procedure is illustrated in the four sketches in Fig. 9. The incisions in the meatal flap should not be made until all pathology has been removed, because the flap must be tailored to suit the needs of each individual case. The illustrations indicate a method of incisions when the attic perforation is small and anterior. If the anterior recess of the attic is large, incision No. 3 should be placed more toward the anterior surface of the external meatus, in order to provide sufficient skin to seal off the middle ear completely. If a small attic fistula remains after surgery, the patient will have drainage after each cold and will be dissatisfied with the surgery because his ear still drains. Some of the failures to obtain a dry ear may be due to this fact. The hearing after surgery may not reach its optimum level unless the tympanic cavity is entirely sealed off.

There are two basic steps in carrying out this procedure: first, the posterior margin of the pars tensa together with several mm. of adjacent meatal skin should be mobilized and displaced medially to provide contact between the pars tensa and the stapes. If the chorda tympani prevents suitable contact, it may need to be sacrificed; the second step is to seal off the tympanic cavity from the attic and antrum with a rotated pedicle skin flap as indicated. If the perforation is more posterior in position than indicated in the sketch, the pedicle of the flap may be created antero-superiorly by varying the incisions accordingly. Modifications must be made to suit each individual case. Occasionally both an anterior and posterior flap may be necessary, with approximation of the two flaps in the mid-attic area. I believe that the actual margin of the attic perforation should be excised in order to eliminate any irregular edge of squamous epithelium which might become buried beneath the edge of the flap and be a nidus of a future cholesteatomatous cyst.

* This term was suggested by Dr. Kurt Tschiassny, Cincinnati, Ohio.

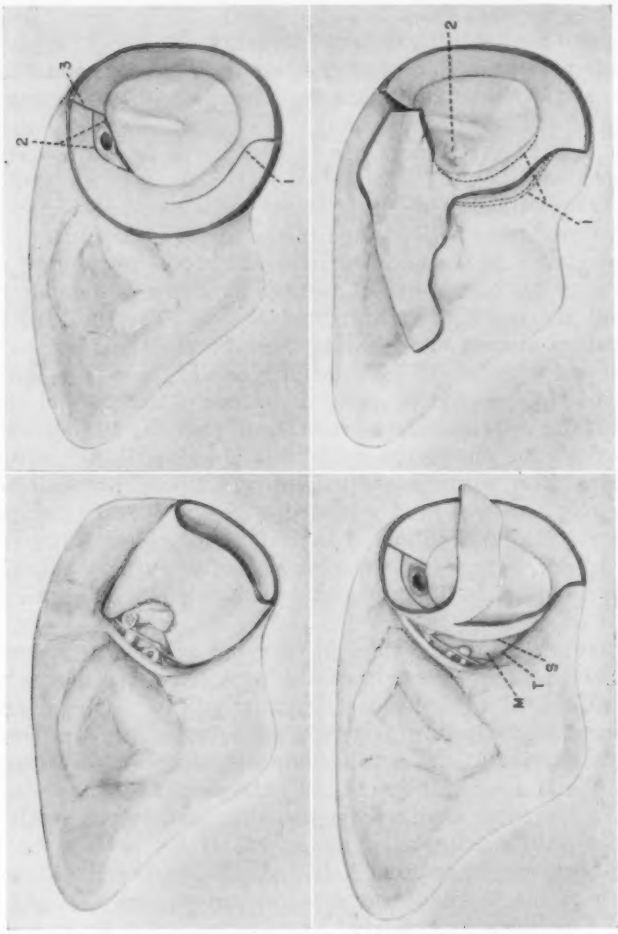

Fig. 9. These are photographs of colored sketches drawn from wax models to represent the four essential steps of myringostapediopexy.

A.—All attic pathology, incus and head of the malleus have been removed. The margin of the original attic perforation is shown at the medial edge of the reflected meatal lining.

B.—Meatal incisions necessary to mobilize flap to permit sealing off of the tympanic cavity from the attic. Incision 3 may be placed more anteriorly if more skin is needed in the anterior attic area. The margin of the perforation is excised by incision 2.

C.—The tympano-meatal flap is freed from the posterior tympanic ring. M.-mucosa on promontory. T.-posterior tympanic ring. S.-posterior edge of tympanic membrane and adjacent meatal skin separated from tympanic ring.

D. Mobilized meatal flap rotated to seal off tympanic cavity. Posterior tympano-meatal flap displaced medially from original position (1) to establish contact between pars tensa and stapes at (2).

If a surgeon prefers, he may elect to use a skin graft to seal off the tympanic cavity instead of a pedicle flap. The disadvantage of using a graft for this purpose is that a failure of the graft to take would result in an attic fistula, and a secondary closure would be necessary if an ideal result is to be obtained.

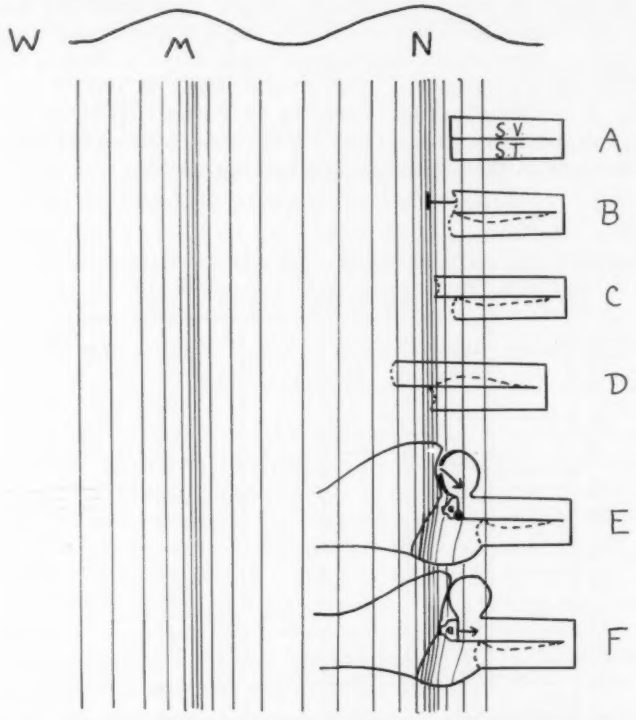

Fig. 10. One complete cycle of sound pressure is shown in the usual manner in W. Pressure peaks are at M and N. Below are drawn the pressure peaks in a frontal manner. Bekesy's box representations of the cochlea have been modified and adapted to show the probable mechanics of the cochlear fluid movement incidental to the application of sound pressure.

A.—Pressure at the two cochlear windows is equal and consequently there is no displacement of the basilar membrane.

B.—A conduction mechanism—represented by the T—against the oval window produces a downward basilar membranes displacement when the pressure peak hits the membrane of the conduction mechanism.

C.—Difference of sound pressure at the two windows causes displacement of the basilar membrane.

D.—The same cochlea a moment later with a reversal of pressure relationship.

E.—Diagrammatic sketch of a fenestrated ear. The pressure peak displaces cochlear fluid inward at the fenestra—indicated by arrow.

F.—This indicates the relationship established in a myringostapediopexis or in an ear on which the Pattee operation has been performed. The incus is absent and the pars tensa (or skin graft) is in contact with the stapes.

A diagrammatic representation of the probable mechanics of such a reconstructed middle ear is shown in Fig. 10. Davis and Walsh[4] have found that the best air conduction level obtainable after fenestration is 20 decibels below the bone conduction threshold. An analysis of the hearing results obtained in patients on whom a modified radical mastoidectomy has been performed indicates that if the incus is absent and there is contact between an intact pars tensa and the stapes, the air-bone gap (unrestored conductive residue) is also 20 decibels. A similar situation is created by Pattee's operation for congenital atresia. Apparently the ossicular chain adds about 20 decibels to the air conduction hearing acuity.

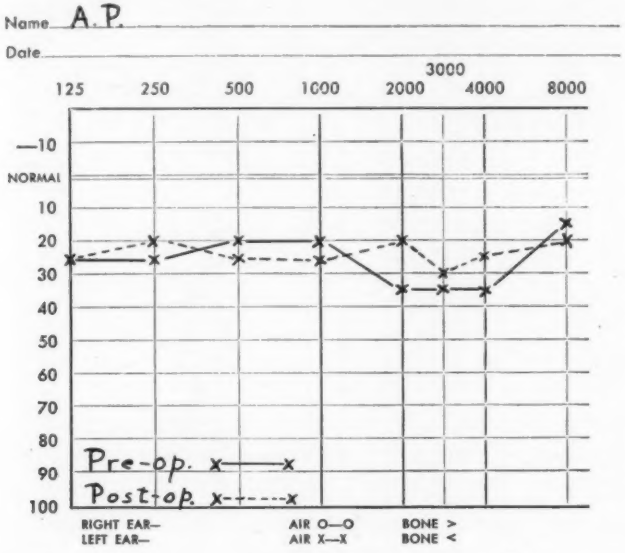

Fig. 11.

Fig. 11 shows the audiograms of a patient on whom a myringostapediopexis was carried out. After removing the attic cholesteatoma some granulations were noted on the head of the malleus. Consequently the incus and malleus head were removed. A serviceable postoperative level of hearing was obtained.

SUMMARY AND CONCLUSIONS.

1. In properly selected cases, a modified radical mastoidectomy (atticomastoidectomy, Bondy mastoidectomy) enables the surgeon to preserve serviceable hearing, and at the same time eliminate pathology originating in the attic.

2. The following prerequisites are essential for a good hearing level after surgery:

a. An air-containing inflatable space over the round window.

b. An intact pars tensa.

c. No fistula from the tympanic cavity into the attic.

d. An intact, or unimpeded ossicular chain

or

if the incus is removed, contact between the pars tensa and the stapes should be assured. If such contact exists before surgery this should not be disturbed.

If such contact is not present, it should be established by the surgical procedure described, *i.e.*, myringostapediopexy.

3. If there is considerable conduction deafness before surgery, the incus and malleus head should be removed and myringostapediopexy accomplished. If serviceable hearing is not restored after complete healing takes place, a secondary fenestration might be considered at a later date if the need for serviceable hearing in the ear in question is sufficient.

4. Each case is an individual problem, and the surgical management is dictated by a thorough study of the hearing before surgery and careful observation of the pathology and status of the ossicular chain during surgery.

BIBLIOGRAPHY.

1. Baron, Shirley: Modified Radical Mastoidectomy; Preservation of the Cholesteatoma Matrix; A Method of Making a Flap in the Endaural Technique. *Arch. Otolaryngol.*, 49:280-320, 1949.

2. Moore, Jas. A.: Endaural Modified Radical Mastoidectomy. The Laryngoscope, 63:113-127, 1953.

3. Juers, Arthur L.: Modified Radical Mastoidectomy Indications and Results. *Arch. Otolaryngol.*, 57:245-256, 1953.

4. Davis, Hallowell, and Walsh, Theo.: The Limits of Improvement of Hearing Following Fenestration Operation. The Laryngoscope, 60: 273-295, 1950.

RECENT TRENDS IN THE MANAGEMENT OF CANCER OF THE MOUTH.*†

RUDOLF J. NOER, M.D.,

(By Invitation).

Louisville, Ky.

Cancer of the mouth may, for convenience, be discussed under three distinct areas: the lip; the tongue, floor of the mouth and gingivae; and the tonsil and oropharynx. Of these, cancer of the lip is the most common and the least dangerous of all. It tends to be of a relatively low grade of malignancy, metastasizes relatively late and then, as a rule, only within the cervical lymph node areas. It has been stated on good authority that not more than 10 per cent of the cancers of the lip will ever metastasize beyond the neck, even in untreated and fatal cases.

In years past it was commonly said that the small primary cancer of the lip could be treated equally well by irradiation, by surgical excision, or by local destruction; the latter, a type of therapy formerly much in vogue among dermatologists. Today we can still say with complete justification that either irradiation or surgery will cure the small and early lesion. Though our radiologic friends may not agree, it is my firm conviction that the larger and more bulky lesions can be excised and immediate repair carried out with less ultimate scarring than is the case if they are destroyed by irradiation. One cannot eradicate malignant tissue by any method without a need for replacement of such tissue, if the lesion be large. I personally prefer to carry out reconstruction at once, with normal tissue, rather than following irradiation, though recognizing full well that modern radiotherapeutic techniques are vastly more effective and less destructive to normal tissue than was the case only a few years ago.

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* Read before the Southern Section, American Laryngological, Rhinological, Otological Society, Inc., Louisville, Kentucky, January 16, 1954.

† From the Department of Surgery, University of Louisville School of Medicine, Louisville, Kentucky.

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So-called "prophylactic neck gland dissection" which was ardently advocated, in fact even insisted upon by many a few years ago, has now largely given way to the more conservative point of view that if the patient can be seen at frequent intervals it is safe to withhold neck dissection unless or until the appearance of palpable nodes indicates that it will be necessary. A considerable portion of these cases will be cured without ever requiring neck surgery.

A word should be said at this point about our present concept of what constitutes an adequate neck node dissection. Formerly, it was a common practice to leave the jugular vein and the sterno-mastoid muscle intact while attempting to remove the cervical lymph nodes surrounding these structures. It is now generally accepted that an adequate block resection of the cervical nodes can be carried out only if these structures, and perhaps other important ones which might be involved, are also removed. One should not hesitate to remove whatever structures are necessary to obtain adequate excision of all involved nodes.

For example, the former viewpoint that sacrifice of both jugular veins in bilateral neck node dissections might be hazardous, has now given way before the weight of evidence of many patients who have had simultaneous radical resections on both sides of the neck at one or two sittings without serious physiologic disturbance. Likewise, we no longer feel that the presence of carcinoma in two noncontiguous triangles of the neck represents a contraindication to radical neck node dissection. The entire problem of neck dissection and factors which influence it has been excellently stated by Hayes Martin and his associates.[1] Probably better than anything else, this paper presents the viewpoint which a large number of us hold today, that neck node dissection need not be done in all cases of lip cancer but in only those where palpable lymph nodes develop, but that where such dissection is to be done it must be a more radical procedure than has been the custom in the past.

Cancer of the Tongue and Floor of the Mouth: This is one of the most malignant of all the mouth carcinomas with which we have to deal, and it is the one in which metastasis to the cervical lymph node areas occurs most directly and most

early. Surely it is one of the areas in which experience with past programs of treatment has been most disappointing. Some idea of the change in approach to the treatment of cancer in this area is apparent from the statement made in 1859, just short of 100 years ago, by Samuel D. Gross, one of the ablest surgeons of his day, in the second edition of his "System of Surgery":

> "The treatment of this affection (carcinoma of the tongue) has generally, at least until lately, been by ablation, either by the knife or ligature. The utility of such proceeding, however, admits of great doubt, and my own opinion has long been that the less we interfere with the disease in this way the better. The results of experience are certainly corroborative of this conclusion . . . . . With judicious management, it is astonishing how long, in many cases, the disease may be kept in abeyance and the final issue warded off"[2].

It appears quite obvious that Dr. Gross must have been sadly discouraged by results of most of the excisional types of therapy which had been followed up to this point; however, it was not long before more radical attempts were made to cure oral cancer and before the modern approach to the so-called "cancer field surgery," including both primary and metastatic nodes in one area appeared. Kocher, in 1880, reported resection of the tongue for cancer, carried out through the submaxillary triangle, together with removal of cervical lymphatic and salivary gland tissue. He later proposed that it might be worthwhile regularly to remove cervical lymph nodes. In 1894, Halsted[3] reported a considerable series of radical mastectomies, emphasizing the en bloc removal of the primary lesion, together with all adjacent lymph node areas. Miles[4] in 1908 described a similar technique for resection of carcinoma of the rectum. Despite general acceptance of the importance of such resection of carcinoma and nodes in continuity, until recently there was a persistent tendency to ignore this principle in treating oral cancer and to deal with the primary mouth lesion, and the lymph nodes to which it might be tributary, as separate and distinct problems.

Increasing perfection of irradiation therapy during the third and fourth decades of this century led many to think in terms of irradiation for the primary lesion and, if it were controlled, later excision of the cervical lymph nodes. Irradiation was also attempted for the neck areas, but there has been increasingly wide acceptance of the fact that this is

rarely a successful means for eradicating carcinoma in the cervical nodes, while a properly performed radical lymph node dissection may be expected commonly to accomplish this purpose; however, even then, there remains an area between the primary lesion and the neck which is not reached by either method, and which is a potential source for subsequent development of metastatic cancer. A dramatic demonstration of this deficiency of the "two-stage" manner of dealing with these lesions was seen in a tumor clinic which I attended elsewhere about three years ago.

A patient with a small proven primary carcinoma of the tongue underwent an adequate hemiglossectomy. He subsequently had a complete radical neck node dissection and none of the nodes showed carcinoma. The man failed to keep in touch, but returned something over a year later with a large, fixed, deeply infiltrated carcinoma lying within the floor of the mouth in the area between the sites of resection of tongue and nodes. It is seldom justifiable to draw conclusions on the basis of one case, but I can think of no more dramatic argument in favor of a resection of carcinoma and nodes in continuity, at one sitting, than the demonstration of what happened to this patient in whom lymphatic tissue was allowed to remain between the primary and the neck lymphatics, both of which were adequately excised.

Increasing dissatisfaction with irradiation therapy for the cervical areas and difficulty with later resection in these areas due to deep fibrosis and scarring, has led many to feel that radiation should not be used in treating the cervical nodes. Concomitantly with this, we find the scope of surgery increasing in all areas of the body. A better understanding of the physiologic needs of the patient, tremendous advances in anesthesia and a wider use of blood have all extended operative indications tremendously in the last 10 or 15 years. The advent of antibiotic therapy has decreased the hazard of infection, which had been one of the chief difficulties with radical surgery.

These factors, together with newer techniques for resection and reconstruction, have made it possible as never before, to apply the accepted principles of cancer surgery to cancer of the mouth. Radical resection of the primary lesion in contin-

uity with its tributary lymphatic areas has thus developed as a wholesome trend in the present day approach to carcinoma of the mouth.

Techniques for this procedure are well recognized and have been adequately described by several authorities.[6,7,8] As a general rule proximity of these lesions to the mandible will require resection of varying amounts of the mandible itself, including the periosteum. There are occasional cases in which it is justifiable to leave the mandible if it is possible to do so with at least a one-centimeter margin between it and the edge of the lesion. Under these circumstances it should be remembered that there is good experimental evidence that the lymphatics connecting the tongue and floor of the mouth with the neck, pass through the mandibular periosteum; thus even though one resorts to the "pull-through" type of operation, it is important to include the periosteum of the mandible in the resection.

As a general rule, it is wise not to lean too far on the side of conservatism but to resect a segment of the mandible whenever there is any question about the wisdom of the less radical procedure. The described operation of resection in continuity has so far shown very promising results with a higher proportion of cure rates than the older procedure of treating the tongue and the lymph node areas as two separate problems ignoring the tissues between them.

Carcinoma of the tonsil and oro-pharynx, the third type to be considered in this discussion, is probably one of the most unsatisfactory problems in all cancer treatment. The area is inaccessible; the lesions are apt to be of a high degree of malignancy, and their inaccessibility makes them difficult for treatment by either irradiation or resection. Because of the great technical difficulty in obtaining a wide excision of these lesions, there are many who feel that most of them are better handled by irradiation, followed thereafter by radical lymph node dissection in the neck. The answer to the best treatment of this cancer, so far as I am concerned, is still not at hand. It is my own personal feeling, and I believe that of the tumor clinic with which I am most frequently associated, that most of these lesions are better handled in the

manner I have described; that is, by irradiation of the primary lesion and radical resection of the cervical lymph nodes.

One should bear in mind, however, that the same type of approach which I have described for resection of the tongue, mandible and neck nodes, can also be extended posteriorly to include the oro-pharynx and tonsillar areas without too great mutilation of the patient. Adequate excision requires a more formidable and a more mutilating operation than the described *en bloc* resection for cancer of the tongue and floor of the mouth. This is, however, sometimes justifiable in late or extensive lesions, or in those which have proven resistant to radiation.

A short film from the University of Louisville teaching files will illustrate better than any words of mine, the type of operation which appears to offer most hope to the patient with oral cancer.

The patient also illustrates the problem which has recently received some attention under the term "multi-centric origin" of cancer, or "field cancer."

CASE REPORT.

A. G., a colored male, age 47, was first seen in November, 1949, at which time biopsy revealed a small squamous cell carcinoma of the tongue to be present in an ulcer which was 3 x1½ centimeters in size in the anterior portion of the tongue adjacent to the floor of the mouth. A hemiglossectomy, hemimandibulectomy, and radical neck node dissection in continuity were carried out. (A tracheotomy was performed as part of this procedure.) He was discharged from the hospital in December, 1949. The patient remained well for two years and was repeatedly observed in the tumor clinic during that period. He was readmitted in April, 1952, at which time he had a small lesion at the base of the tongue on the left side, which proved to be an infiltrating carcinoma. This was treated by X-ray therapy since it was a small area quite posteriorly situated, and one which it was felt would not require a second radical procedure like the one done on the right.

In March, 1953, there was a small area of thickening at the anterior portion of the tongue; biopsy revealed this to be an *in situ* carcinoma which had been excised for biopsy. The patient was readmitted in April, 1953. At that time he gave no evidence of any recurrence anywhere in his mouth or neck; however, he was having difficulty in swallowing and an esophagoscopy revealed a squamous cell carcinoma of the esophagus, histologically Grade II. An exploratory thoracotomy was carried out, and the carcinoma of the esophagus was found to involve the trachea and to be non-resectable. A gastrostomy was carried out, but the patient died of carcinoma about two months thereafter. Autopsy revealed the carcinoma of the upper end of the esophagus, which had perforated into the trachea, together with another ulcerative lesion at the lower end of the esophagus which showed *in situ* carcinoma.

In summary, then, this patient had three separate infiltrating carcinomas, all believed by the pathologist to be primary: in 1949 of the right tongue, in 1952 the left tongue adjacent to the tonsilar fossa, and in 1953 at the upper end of the esophagus. He also had two other areas from which diagnoses of *in situ* carcinomas were returned, and which were also felt to be independent in origin: in 1953 the left tongue, anteriorly; in 1953 the esophagus, 2½ centimeters from the cardia. Thus, this patient, though cured of his first carcinoma by a radical resection, subsequently died of an independent carcinoma of the esophagus.

CONCLUSIONS.

1. Lip cancer may be eradicated by irradiation or surgical excision. If the patient develops palpable cervical lymph nodes, a truly radical cervical node dissection will be required on one or both sides.

2. Most cancer of the tongue and floor of the mouth is best handled by a resection of the primary lesion in continuity with a radical neck node dissection.

3. Carcinoma of the tonsil and oro-pharynx is a problem not yet solved. At present we favor irradiation of the primary followed by radical cervical node dissection extending up to the irradiated area. Occasional cases may require wide resection including the mandible, floor of the mouth and cervical node areas, an extension of the operation described for tongue and mouth cancers.

BIBLIOGRAPHY.

1. Martin, Hayes; Delle Valle, Bernardo; Ehrlich, Harry; and Cahan, William G.: Neck Dissection. *Cancer*, 4:441-499, 1951.

2. Gross, Samuel D.: "System of Surgery," 2nd ed., Blanchard and Lea, Philadelphia, 1859.

3. Kocher, Theodore: Ueber Radicalheilung des Krebses. *Deutsche Ztschr. f. Chir.*, 13:134-166, 1880.

4. Halsted, William S.: The Results of Operations for the Cure of Cancer of the Breast Performed at The Johns Hopkins Hospital from June, 1889, to January, 1894. *Ann. Surg.*, 20:497-555, 1894.

5. Miles, W. E.: A Method of Performing Abdominoperineal Excision for Carcinoma of the Rectum and of the Terminal Portion of the Pelvic Colon. *Lancet*, 2:1812-1813, 1908.

6. Sugarbaker, E. D., and Gilford, J.: Combined Jaw Resection-Neck Dissection for Metastatic Carcinoma of Cervical Lymph Nodes Secondarily Involving the Mandible. *Surg., Gynec. and Obst.*, 83:767-777, 1946.

7. Slaughter, D. P.; Roeser, E. H.; and Smejkal, W. F.: Excision of the Mandible for Neoplastic Diseases: Indications and Techniques. *Surg.*, 26:507-522, 1949.

8. Ward, Grant E., and Robben, John O.: A Composite Operation for Radical Neck Dissection and Removal of Cancer of the Mouth. *Cancer*, 4:98-109, 1951.

SMOKER'S LARYNX.*†

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Entertainment today is being provided by television and radio to an ever increasing degree. Cigarette companies are among the biggest sponsors. Smoking is often portrayed as the mark of a blase and sophisticated individual, constituting a strong attraction for adolescents. The commercials constantly extol the joys of smoking and the absence of any harmful effects. These claims are often bolstered by the reports of surveys of physicians who have found no adverse effect of smoking. Even "Nose and Throat specialists" are included as certifying to the absence of any irritating qualities in a particular brand of tobacco. This type of advertising must be disturbing to laryngologists who so frequently see throat conditions which they attribtute to smoking and who attempt to convince the patient to stop the habit.

This presentation aims to review the diseases of the larynx that are considered to be the result of smoking and to evaluate the evidence that serves to indict tobacco. The laryngeal conditions most often blamed on smoking are: *1.* chronic nonspecific laryngitis; *2.* polypoid degeneration of the vocal cords; *3.* leukoplakia or keratosis, and *4.* carcinoma.

CHRONIC NONSPECIFIC LARYNGITIS.

This is an exceedingly common disease. Numerous factors may contribute to its development, such as dusty air, abuse and faulty use of the voice, infection of the sinuses or the chest, with chronic cough, mouth breathing, alcohol, as well as tobacco smoke. Actually, in routine office practice the condition is so commonly seen in heavy smokers that this seems the single most important and common etiologic agent.

The gross appearance of the larynx is characteristic. There is hyperemia of the mucosa so that it is darker red and furry

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in appearance. Thickening of the mucosa is evidenced by a rounding of the arytenoids and blunting of the sharpness of the landmarks. The smaller capillaries may be dilated. The true cords do not appear as white, flat bands, but are rounded, thickened, and hyperemic, with visibly dilated vessels. The edges may be irregular with sticky adherent secretion.

The patient may not be aware of an alteration of the voice, or of hoarseness, or complain of any symptoms referable to the throat. Chronic laryngitis is a common incidental finding in the routine examination of heavy smokers. When there are no subjective complaints, the chief significance of its existence would seem to be in the possibility of chronic laryngitis acting as a precursor of more serious disease. Speaking with chronically inflamed vocal cords may lead to voice strain that results in the formation of polyps or of polypoid degeneration. The prolonged irritation of the mucosa may cause keratosis or even malignancy.

The evidence that smoking is an important etiologic factor in chronic laryngitis is based on: *1.* the conclusions of most clinicians with the widest experience; *2.* the well established fact that the condition is usually reversible; when the irritation of smoking is removed, the larynx often returns to normal.

POLYPOID DEGENERATION OF THE CORDS.

In 1950 Myerson[1] described this lesion of the vocal cords as a clinical pathological entity directly due to smoking. His conclusions were based on the study of 143 patients with edema or diffuse polypoid changes of the anterior two-thirds of the true vocal cords. His illustrations reveal five different stages, from simple edema to bilateral rounded polypoid masses with a sessile base but freely movable because of their thin attachment. He described the histology as an edematous fibroma. The epithelium may vary somewhat, but usually is the normal stratified type of the free edge of the cord. The bulk of the tumor consists of fibrous tissue in an edematous stroma. Myerson postulates that the local irrita-

tion of smoking first results in an outpouring of serum into Reinke's space. As a result of this chronic edema there is increased fibrosis, and finally a polypoid tumor develops.

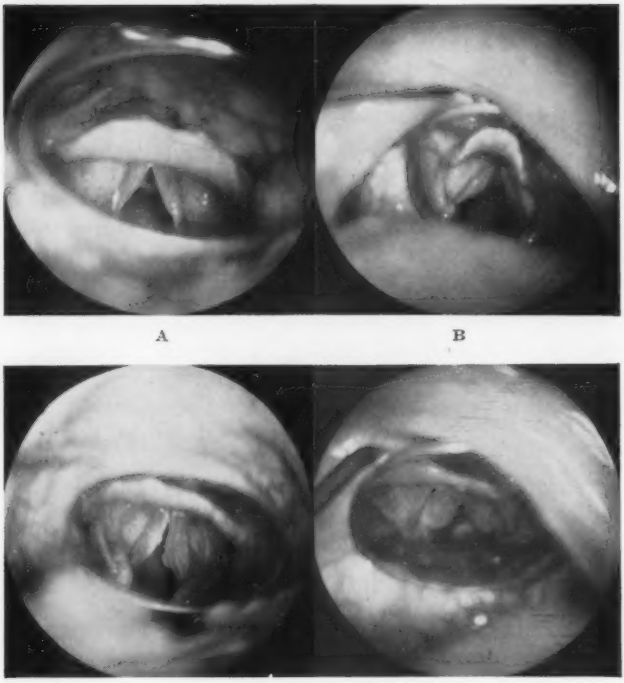
His evidence for concluding that this polypoid formation was directly due to smoking was: *1.* all of his 143 patients were heavy smokers; *2.* his observation that in the early stages when only edema was present the swelling would disappear within 24 hours when smoking was stopped; *3.* his histologic studies of the various stages in the development of these growths.

Jackson[2] termed this condition polypoid corditis. He described it as a frequent finding in chronic laryngitis. He thought these tumors were of inflammatory origin, and described them, along with pedunculated polyps, as edematous fibromata. Holinger[3] described this condition with the term polypoid degeneration of the cord, under the classification on non-neoplastic tumors. He reported on 112 patients with this disease, and found the histology similar to predunculated polyps.

Putney[4] and Clerf called the disease a chronic hypertrophic laryngitis and concluded that it was due to vocal abuse or infection. They advised stripping of the cords. They also found the chief histologic alterations as edema and fibrosis.

Since Myerson's report, twelve patients with polypoid degeneration of the cords have been studied. Eleven were smokers; one had never smoked but had used snuff for many years. The gross appearance was as described by Myerson (see Figs. 1, A, B, C, D, 2C). In five of the patients the growths were removed, and examined microscopically. The findings were essentially as described above. On comparison with sections made of pedunculated polyps, a similar histopathology was manifest (see Fig. 2, A, B, D, C).

Removal of the growths was not advised in these patients until the patients had given up smoking. In one patient this resulted in a complete disappearance of large bilateral polypoid masses. The improvement did not occur rapidly, within 24 hours as described by Myerson, but took place gradually over several months.



A B C D

Fig. 1.—A. Photograph of the larynx of a male excessive smoker revealing diffuse edema of the anterior half of both cords.

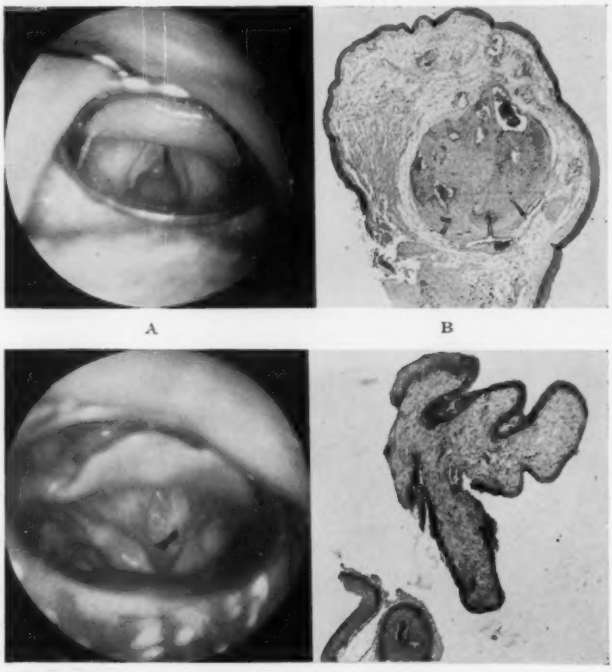
B. Polypoid degeneration of the right cord of a female non-smoker who had used snuff for many years.

C. Male heavy smoker and constant talker. There is polypoid degeneration of both cords, with superficial leukoplakia of the right.

D. Marked polypoid degeneration of the anterior half of both cords in a male excessive smoker.

REPORT OF A CASE.

A 72-year-old male executive was seen because of hoarseness following a cold of 10 days' duration. He had frequent episodes of hoarseness treated by local applications, had been a heavy cigarette smoker for many years, and talked a great deal over the long distance telephone as well as in conferences. Mirror laryngoscopy revealed marked polypoid degeneration of the anterior two-thirds of both true cords. The masses were pale and edematous in appearance. They were so large they occluded the anterior portion of the glottis, but were very mobile, moving up and down on respiration. There was diffuse hyperemia of the remainder of the larynx. The condition was described to the patient and he was urged to stop smoking, which he did. Within two weeks there was considerable decrease in the size of the ploypoid enlargements. After three months' observation, the cords were normal in appearance and the patient's only complaint was his weight gain since stopping smoking.



A B

C D

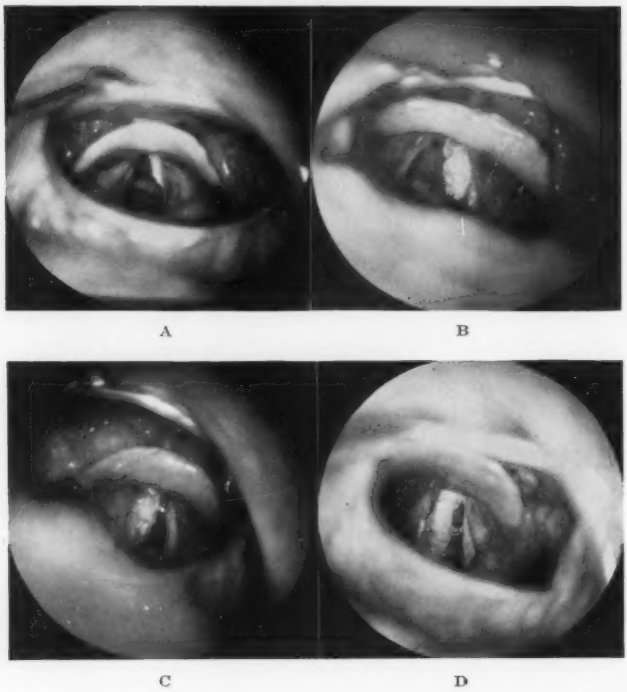
Fig. 2.—A. Typical pedunculated polyp of the right cord.

B. Photomicrography of the polyp removed from the patient in 2.—A. There are only minor alterations of the mucosa. The bulk of the tumor consists of an edematous stroma with fibrosis. There is a large round area of hyaline.

C. Male excessive smoker, drug addict and constant talker. There is marked polypoid degeneration of the right cord.

D. Photomicrograph of the tissue removed from the patient. 2.—C. There is thickening of the epithelium. The stroma is edematous with dense fibrosis. (Compare with 2. B).

Pedunculated polyps (see Fig. 2A) of the vocal cords are known to occur in non-smokers. Their formation is usually attributed to vocal abuse. Diffuse polypoid degeneration of the cords has a similar histopathology, and it would seem inconsistent to consider them due to smoking and the discrete polyps to vocal strain. The similar histopathology suggests a similar etiology. This common denominator may well be vocal strain, whether from vocal abuse, or from speaking with the constantly inflamed cords of chronic laryngitis due to



A B C D

Fig. 3.—A. Male heavy smoker with keratosis of the anterior half of the left cord.

B. Marked keratosis of the left cord of a male excessive smoker.

C. Keratosis and infiltration of the right cord of a male excessive smoker. Biopsy: Carcinoma arising in leukoplakia.

D. Keratosis and infiltration of the right cord of a male heavy smoker. The right cord had been stripped of leukoplakia six years previously. Biopsy: Carcinoma.

smoking. Friedberg[5] and Segall studied Reinke's space and its relation to cordal polyps. They explain the usual site of discrete polyps at the junction of the anterior and middle thirds as due to the existence of a "vibration node" at this site. If the outpouching does not occur at this point, a sessile type of polyp results. This concept does not detract from the importance of smoking in the etiology of polypoid degeneration, but rather explains that it is not necessarily a specific effect.

KERATOSIS, HYPERKERATOSIS AND LEUKOPLAKIA.

These terms are used synonymously by many authors and as distinct entities by others. Jackson[2] and Jackson describe leukoplakia and keratosis as separate diseases. New[16] and Erich consider them as distinct though similar conditions. Clerf,[7] Putney[8] and Holinger[3] use the two terms as indicating the same disease. As used in this discussion, the term keratosis denotes the chalky white localized elevations of the mucosa, which histologically reveal benign epithelial hyperplasia, without invasion of the submucosa (see Fig. 3, A, B). There is usually an associated chronic laryngitis, causing the whitish areas to contrast all the more with the hyperemic mucosa. A chronic irritation is usually advanced as the reason for the development of keratosis, with smoking listed as the commonest offender. Clerf[7] states that he is convinced that smoking is a predisposing factor, but admits it has not been proven. He is impressed with a deficiency of Vitamin A as an additional factor in some patients. Putney[8] and O'Keefe state that smoking has long been considered a salient factor. In their series of 125 patients with keratosis of the larynx only 11.2 per cent were in non-smokers, compared with 34 per cent of non-smokers in the general population. LeJeune[9] believes that keratosis is being seen more commonly these days. All of his patients with the disease used cigarettes excessively. In a recent study by Friedberg[10] and Wallner, all patients with benign keratosis were heavy smokers.

REPORT OF A CASE.

A 49-year-old male was referred for examination of the sinuses as a possible cause of a chronic cough. He complained of postnasal discharge and hoarseness for three weeks. He admitted smoking two packs or more of cigarettes daily. On examination the nasal airways were adequate, the mucosa was quite hyperemic. The sinuses were moderately clear on transillumination and X-rays revealed them to be normal. The tonsils had been cleanly removed. The larynx revealed diffuse leukoplakia of the anterior third of each true cord. There was intense hyperemia of the rest of the larynx. The patient agreed to stop smoking, and no other treatment was given. In three weeks he reported his voice greatly improved and less postnasal discharge. The leukoplakia was less marked. Two months later the leukoplakia had disappeared and only a slight hyperemia remained. Six months after the first examination he reported that his cough and postnasal discharge had completely disappeared and that his voice was normal. His larynx appeared normal.

We may summarize the evidence that smoking is an important factor in the etiology of keratosis: *1.* this is the con-

clusion of all writers, based on their clinical experience, *2.* keratosis is often reversible in that when smoking is stopped, the condition may disappear, or is less likely to recur after removal.

CARCINOMA.

The etiology of cancer remains an enigma, but chronic irritation is frequently listed as an important factor. It is a common observation that malignant disease of the larynx is often preceded by long periods of hoarseness. Jackson[2] states "Precancerous conditions are present in probably 75 to 80 per cent of the cases . . . as a clinical fact we have rarely found cancer developed in a normal larynx." Szanto, a pathologist, writing in Lederers'[11] text says, "Cancer rarely develops in a normal larynx . . . in at least half of the cases of laryngeal cancer, precancerous lesions antedate the malignant growth" (see Fig. 3 C). "Many writers have emphasized the importance of precancerous lesions in the development of laryngeal carcinoma. Precancerous and malignant growths may have similar gross appearance, they may be difficult to differentiate histologically, and a lesion that was regarded as benign may later become malignant." Clerf, Putney and O'Keefe, and New and Erich describe observing patients with benign keratosis for long intervals and finally finding evidence of carcinoma (see Fig. 3 D). Friedberg[10] and Wallner found of 39 patients with cordal cancer, 19 or 61 per cent had leukoplakia of the vocal cords.

It thus becomes obvious that if we accept smoking as an etiologic factor in keratosis, we must also regard it as a factor in the development of cancer. Even in those patients with laryngeal cancer without known precancerous lesions, there is evidence that heavy smoking plays a role. In the series mentioned, studied by Friedberg[10] and Wallner, of 116 patients with laryngeal cancer, there were 97 known smokers, 3 non-smokers, and in 16 the information was not available. This incidence of smokers is appreciably higher than the 73.7 per cent figure quoted by Wynder[12] and Graham as representing the smokers in a general (non-cancer) population.

There have been a number of other careful statistical surveys on the relation of smoking to cancer of the respiratory

tract.[13,14] These reveal a significantly higher percentage of smokers among those with cancer than among the control groups.

The evidence that smoking is a factor in the development of laryngeal cancer is circumstantial. It is based on the judgment of most clinicians with the widest experience, who have observed the high percentage of smokers among patients with laryngeal malignancy.

TREATMENT.

One of the fundamentals in the management of chronic laryngeal disease is for the patient to stop smoking. This seems a simple enough remedy, but in practice it is most difficult to accomplish. Most patients will offer to "cut down" on their consumption, or to stop for awhile. The prospect of permanently abolishing the habit seems to appall them. It is a question whether it is better to insist on complete abstinence, with less chance of success, or to compromise and permit a greatly curtailed amount of smoking, as described by Jackson.[2] Obtaining the patient's cooperation is certainly a difficult problem, taxing the physicians' power of persuasion, salesmanship and patience. It requires a great deal more time and effort to convince the patient of the necessity of not smoking than to administer some local treatment. The physician's efforts may be rewarded by observing the improvement in the larynx, and in the gratitude expressed by the patient for his general improvement and sense of well being.

COMMENT.

This review indicates that there is no one disease of the larynx that has been proven as specifically and entirely due to smoking. Rather, excessive smoking may be considered the chief, though not the only factor in three diseases, and probably important in the fourth. Based on frequency of occurrence, chronic laryngitis could be considered as the typical "smoker's larynx," though keratosis is more likely to be thought pathognomonic of excessive smoking.

One may ask, what would constitute definite proof of the etiology of a disease? Animal experimentation gives only cor-

roborative evidence, though the results of the work of Wynder,[15] Graham and Croniger with tobacco tar producing skin cancers in mice seem as applicable to cancer of the larynx as of the lung. The fact that a disease may have more than one cause is not proof against any given agent. The best evidence we have at present is clinical, the fact that we can often reverse the disease process when the irritant is withdrawn. This is true for chronic non-specific laryngitis, and to a lesser degree for leukoplakia and polypoid degeneration of the cords.

Accurate statistics are not available, but chronic non-specific laryngitis is certainly very common in individuals who have smoked excessively for years. The patient may not be aware of any abnormalities in his voice and have no throat complaints. Alterations in the voice may not be as noticeable in men as in women, where a huskiness or lowering of pitch is more obvious and objectionable.

Considering the number of persons that smoke, the more serious forms of laryngeal disease are not common. Even if we accept the higher percentage of smokers among patients with laryngeal cancer as significant, it does not tell the other aspect of the question, how dangerous is smoking? Mortality statistics of the U. S. Census Bureau[16] lists 0.76 per cent of deaths of men past 40 due to cancer of the respiratory tract. This would indicate that even if tobacco smoke is a carcinogenic agent it is a weak one. It is doubtful if we are justified in using fear of cancer alone in attempting to curtail a habit that seems to bring so much satisfaction to so many millions. Scientific accuracy would require us to stress instead the less serious but common irritative conditions associated with smoking.

In any discussion of the harmful effects of smoking it must be remembered that the larynx is only one organ that may be adversely affected. To obtain the complete picture, we should consider the effect on the nose; in decreased olfaction, blocking and nasal and post nasal discharge. There may be a chronic pharyngitis with dryness and sore throat. The possible relation to cancer of the lung is receiving wide publicity. The chronic "smoker's cough" is well known. The effects of smoking on the digestive system, heart and circula-

tory systems are more controversial; however, when the overall effects of smoking are studied, deleterious results are not uncommon. Once acquired, the habit usually remains with an individual. Many of the adverse effects are not noticed until after years of smoking. Graham and Wynder[12] found the carcinogenic effect in the lung occurred after 30 to 35 years, or one-half the life span. This was substantiated by their animal experiments.[15] Short term surveys, purporting to discover no ill effects after a few months' observation, are apt to be misleading.

The results of polls of physicians as expressed by the tobacco companies may not be as significant as the conclusions of the smaller number of investigators who have studied the problem more intensively.

SUMMARY AND CONCLUSIONS.

The irritation of tobacco smoke is a frequent cause of chronic laryngeal disease. Smoking is an important factor in chronic non-specific laryngitis, in polypoid degeneration of the vocal cords and in keratosis. Statistical evidence also indicates it is a factor in the etiology of laryngeal cancer.

REFERENCES.

1. Myerson, Mervin C.: Smoker's Larynx. A Clinical Pathological Entity. *Ann. Otol., Rhinol., and Laryngol.*, 59:541-546, June, 1950.

2. Jackson, Chevalier, and Jackson, Chevalier, L.: The Larynx and Its Diseases. 51, 136-146, 141 and 342, 376, 335-336. W. B. Saunders Company. Philadelphia and London, 1937.

3. Holinger, Paul H., and Johnston, Kenneth C.: Benign Tumors of the Larynx. *Ann. Otol., Rhinol., and Laryngol.*, 60:496-509, June, 1951.

4. Putney, F. Johnson, and Clerf, Louis H.: Treatment of Chronic Hypertrophic Laryngitis. *Arch. Otolaryngol.*, 31:925-929, June, 1940.

5. Friedberg, Stanton A., and Segall, Walter H.: The Pathologic Anotomy of Polyps of the Larynx. *Ann. Otol., Rhinol., and Laryngol.*, 50:783-789, Sept., 1941.

6. New, Gordon B., and Erich, John B.: Benign Tumors of the Larynx: A Study of Seven Hundred and Twenty Two Cases. *Arch. Otolaryngol.*, 28:841-910, Dec., 1938.

7. Clerf, Louis H.: Keratosis of the Larynx. *J.A.M.A.*, 132:823-826, Dec., 1946.

8. Putney, F. Johnson, and O'Keefe, John J.: The Clinical Significance of Keratosis of the Larynx as a Pre-Malignant Lesion. *Ann. Otol., Rhinol., and Laryngol.*, 62:348-357, June, 1953.

9. LeJeune, F. E. Surgical Treatment of Early Carcinoma of the Larynx. *Trans. Amer. Laryngol., Rhinol., and Otol. Soc.*, 55:318-325, 1951.

10. Friedberg, Stanton A., and Wallner, Linden J.: Selected Problems in the Diagnosis of Laryngeal Carcinoma. *A.M.A., Arch. Otolaryngol.*, 58: 521-535, Nov., 1953.

11. Lederer, Francis L.: Diseases of the Ear, Nose and Throat. F. A. Davis Company. 6th Edition, 1325, Philadelphia, 1952.

12. Wynder, E. L., Graham, E. A.: Tobacco Smoking as a Possible Etiologic Factor in Bronchiogenic Carcinoma; A Study of 684 Proved Cases. *J.A.M.A.*, 143:329-336, May, 1950.

13. Schrek, Robert; Baker, Lyle A.; Ballard, George P., and Dolzoff, Sidney: Tobacco Smoking as an Etiologic Factor in Disease. I. Cancer. *Cancer Research*, 10:49-58, Jan., 1950.

14. Mills, Clarence A., Porer, Marjorie Mills.: Tobacco Smoking Habits and Cancer of the Mouth and Respiratory System. *Cancer Research*, 10:539-542, Sept., 1950.

15. Wynder, Ernest L.; Graham, Evarts A., and Croniger, Adele B.: Experimental Production of Carcinoma with Cigarette Tar. *Cancer Research*, 13:855-864, Dec., 1953.

16. United States Bureau of Census, Mortality Statistics, U. S. Government Printing Office, 1936.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Registrations must be completed before August 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota.

The Alexander Graham Bell Association for the Deaf (formerly The Volta Speech Association for the Deaf) will hold its 1954 Summer meeting in St. Louis, June 14-18 inclusive. The theme of this meeting is "Let's Face the Issues," and some of the questions to be discussed are: "Where should the deaf child receive his education?" "What are the possibilities and limitations of auditory training?" "What are the principal issues in teaching speech?" and "What contributions can be made to the understanding of the deaf child by persons in peripheral fields?"

All sessions will be held at the Chase Hotel, but there will be Open House at Central Institute for the Deaf and St. Joseph Institute for the Deaf.

For further information, address: Robert Goldstein, 818 S. Kingshighway, St. Louis 10, Missouri.

NON-SURGICAL MANAGEMENT OF MENIERE'S DISEASE.*

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The voluminous literature on the symptom complex of Meniere's disease has not clarified its pathogenesis nor has it positively demonstrated more of the pathology in the inner ear than the dilatation of the endolymphatic duct. Obviously, any plan of treatment of the disease has been and still is empirical. That such empirical management leaves something to be desired is only too apparent in reviewing even the literature of the last few years, and then in applying the suggestions for treatment in one's own practice.

To avoid confusion in terminology and symptomatology with other authors, the following clinical picture has been our basis for a diagnosis of Meniere's disease or "labyrinthine hydrops." These terms are used synonymously for the symptom complex of a low tone perceptive deafness that usually fluctuates widely, at least in the early stages; a roaring type of tinnitus; complaint of a sense of pressure or fullness in the ear, and paroxysmal attacks of vertigo. Hyperacusis not infrequently is present and often the symptom of diplacusis can be demonstrated. This is a relatively easy diagnosis to make, and it is rarely a diagnostic necessity to have the results of such special functional tests of the labyrinth as recruitment and difference limen tests, discrimination scores with the P. B. List, or even caloric tests of equilibrium.

Not all of the preceding symptoms need to be present to justify a diagnosis of Meniere's disease. More often than not the entire symptom complex is present, but less frequently one sees either the picture of cochlear or vestibular symptoms alone.

PATHOGENESIS.

To me the most appealing descriptions of the possible etiology and pathogenesis of Menier's disease, based upon keen

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clinical observations and the application of the knowledge of basic physiological studies of the autonomic nervous system, are those of Hilger[1,2] and Williams.[3,4] Hilger[1,2] represents the various clinical syndromes of inner ear pathology as resulting from autonomic dysfunction in the labyrinthine neurosvascular sphere. This dysfunction produces angiospastic change in the labyrinthine arterial supply and, thus, labyrinthine ischemia with a resultant transudate from ischemic damage to the capillaries. In this way variable cochlear and vestibular involvement can occur, the differences being due to the degree of ischemia and the nature of labyrinthine elements involved.

Williams[3,4] states that a localized autonomic dysfunction producing a stereotyped vaso-spastic vaso-atonic condition in the peripheral vascular bed is the basis for the development of pathologic changes in Meniere's disease. He feels that autonomic dysfunction and allergy are synonyms describing identical phenomena. The arteriolar spasm with capillary and venule dilatation causes anoxia of the capillary loop, injury to the endothelium and increased permeability of the capillary loop. This type of involvement of the stria vascularis results in an increased volume of endolymph with an increased protein content and higher osmotic pressure producing the typical cochlear symptoms. Williams states that the vertiginous crises are produced by vasospasm of the branches of the internal auditory artery supplying the maculae and cristae which causes local anoxia, or by a sudden release of vasospasm producing irritation from a sudden resumption of blood flow.

THERAPY.

Authors have varied considerably in selecting the chief instigating factors of autonomic dysfunction to attack in their rationale for therapy of Meniere's disease. The narrow selection of precipitating factors is exemplified by the approach of such writers as Wright[5] in his focal infection theory; Selfridge[6] and others with their avitaminosis theory, or Gundrum[7] in a recent report suggesting an allergic etiology in 34 of 57 non-traumatic cases of Meniere's symptom-com-

plex. Mygind and Dederding,[8] and Furstenberg and associates[9] were others who gave principal emphasis to one factor, considering respectively the water balance or the sodium retention as the single most important factor to be controlled in the treatment of Meniere's disease.

My own feeling is that numerous factors must be considered as possible precipitators of the crisis of localized autonomic dysfunction in the inner ear. Any one of these, or a combination of several, can be the trigger in a given case. The relative importance of the different factors is difficult to evaluate, but the broader viewpoint of etiology is more in keeping with that expressed by Williams or Hilger.

In attempting to evaluate an individual patient with Meniere's disease we try, through a careful history, to elicit evidence of the following: any emotional or stress influence; tobacco, alcohol, salt, fluid or specific dietary over-indulgence; medications taken over a long period, including hormones and vitamins; coincidence between severe upper respiratory infections or influenza with attacks; apparent variation with menses, season of the year, climate, trips or physical environmental contacts; clinical manifestations of specific antigen-antibody allergy, such as in seasonal or perennial allergic rhinitis, bronchial asthma, eczema or allergic gastro-intestinal disorders, other vascular phenomena such as classical and atypical histaminic head and neck pains.

Any one of the above mentioned factors has been the only important one in one or more cases in our practice, and a combination of several such factors has been even more common; however, unfortunately in our experience, quite frequently none of the factors listed can be proved, or even strongly suspected, in certain individual cases. The most rewarding single etiologic lead which we have been able to establish has been a specific antigen-antibody reaction in approximately 5 to 10 per cent of the cases of Meniere's disease seen in our office in a given year. The most completely unrewarding search has been for foci of infection or vitamin deficiencies.

In outlining our usual medical management of the patient with Meniere's disease, I am concentrating on the office treatment rather than the occasional acute vertiginous or cochlear crisis necessitating hospitalization. As stated earlier, an attempt is made to establish any fairly definite precipitating factors by a thorough history, examination and finally by a therapeutic test. The therapeutic test, in this instance, is the relief of symptoms by the removal of the offending factor or by appropriate treatment of the factor, and the recurrence of symptoms upon re-exposure to the factor, or by stopping treatment. Applying this same approach to establishing specific allergic etiology is the reason for our confidence in the control of 5 to 10 per cent of our cases of labyrinthine hydrops by specific allergic management. The most commonly missed specific allergic factor is apt to be one of the staple foods, unless it is unmasked by specific feeding tests, according to Rinkel's[10] technique.

If we fail to establish or to control the precipitating factors, or if such an attempt is apt to be a long drawn-out procedure, in which eventual success is uncertain, then what I term nonspecific management is begun.

Our modification of the "salt-free" Furstenberg diet is shown in Fig. 1. I find that by liberalizing the diet to allow salted butter and the ordinarily prepared bread, with the use of the proprietary salt substitutes, most patients will adhere to the program. This amounts to approximately a 1.0 gm. sodium diet. To this is added potassium chloride, two teaspoons of a 25 per cent aqueous solution three times daily during meals for three consecutive days, alternated with two days of omission. The employment of "salt-free" diets and ammonium or potassium chloride has become almost a ritual in the management of labyrinthine hydrops, and certainly this ritual needs drastic re-evaluation in the face of the recent report by Perlman and his associates[11] of their complete inability to produce a consistent effect upon cochlear or vestibular function in fifteen patients with active Meniere's disease, while on hospital programs which produced measured increases or decreases of serum sodium levels.

Fig. 1.

FURSTENBERG DIET.

GROUP A—The following foods may be taken daily:

1. Eggs, meat, fish and fowl as desired, without salt.
2. Bread as desired, without salt.
3. Cereal, one of the following: Farina, oatmeal, rice, puffed rice, puffed wheat.
4. Potato and one or more servings of any of the following: a) macaroni, b) spaghetti, c) rice, d) corn, e) cranberries, f) prunes, g) plums.
5. Milk as desired.
6. Vegetables and fruits daily of any fruit and of any vegetables not included in groups "B" and "C" as desired.
7. Butter, cream, honey, jellies, jams, sugar and candy permitted as desired.
8. Tea and coffee as desired.

GROUP B—Foods to be avoided:

All salt meats and salted fish, or bread, crackers and butter prepared with salt. Carrots, clams, condensed milk, raisins, caviar, cowpeas, olives, spinach, cheese, endive, oysters.

GROUP C—Foods to be taken no more than twice weekly:

Lima beans, beets, buttermilk, cantaloupe, cauliflower, celery, chard, dried coconut, dried currants, dates, figs, horseradish, kohlrabi, limes, muskmelons, peanuts, peaches, mustard, pumpkin, radishes, rutabagas, strawberries, turnips, turnip tops, watercress.

NOTE: All Foods to Be Prepared and Served Without Salt.
Water intake unrestricted although excessive quantities of liquid should not be taken.

MEDICATION: Potassium chloride, 2 tsp., 25% solution, t.i.d., alternate 3 days of intake with 2 days of omission.

SALT SUBSTITUTE: Diasal.

Most authors are agreed upon the need of using a vaso-dilating agent to combat the basic functional lesion of vasospasm. The disagreement that exists in this type of therapy is chiefly in the specfic vasodilator used, and somewhat in the route, amount and frequency of administration. Of the smooth muscle dilating agents our preference is histamine. I feel that the effect of histamine is probably secured by its reaction upon the spastic-atonic state of the peripheral vascular bed, rather than by a so-called "desensitization."

We make a consistent attempt to individualize the histamine therapy by varying the dosage according to the patient's reaction or lack of reaction. This is the "small optimum dosage of Hansel" in contrast to the usually reported much stronger increasing dosage technique of subcutaneous administration, or the constant intravenous dosage. The intravenous dosage usually amounts to 1 cc. of 1:1000 dilution of histamine given daily at a slow drip rate. The build-up

therapy consists of twice daily subcutaneous injections of 1:10,000 dilution, up to the 1:1000 dilution. It is unfortunate that the criticisms of the results of histamine therapy are based upon the latter two types of administration, and are often made by the same authors who advocate a small optimum dosage method for giving either subcutaneous monoethanolamine nicotinate (nicamin) or oral nicotinic acid.

The optimum dosage technique as we employ it begins with 0.1 cc. of a 1:100,000,000 dilution of histamine diphosphate solution given subcutaneously. If no noted effect follows within 24 hours then the dosage is increased one dilution to 0.1 cc. of a 1:10,000,000 dilution given within one to three days after the first injection. The subcutaneous histamine dosage is increased in this manner until the optimal dosage, or that amount which will just relieve the symptoms is reached. Once the dilution has been found which will give a day or so of improvement, the dosage is increased by making the amount of that dilution greater; for example, from 0.1 cc. to 0.25 cc. to 0.50 cc., etc. The ultimate best results are found to be on a subcutaneous dosage which maintains the patient symptom-free for intervals of three to seven days, supplemented by sublingual administration of histamine one or two dilutions stronger than the subcutaneous dosage, using two drops twice daily. If the initial dosage of 0.1 cc. of the 1:100,000,000 dilution precipitates a Meniere's attack, then the dosage is lowered to 1:10,000,000,000 and further adjustments are made to obtain an optimum dose.

When the patient under non-specific treatment has been free of vertigo, and the hearing has been stabilized at the highest possible level without the symptoms of distortion and pressure for a period of six to eight weeks, the histamine and potassium chloride are discontinued, and the salt-free diet is liberalized. Any recurrence of symptoms is followed by the resumption of the complete program.

I feel that one can best judge that a given histamine injection has been effective by an almost immediate (within several minutes to a few hours) improvement in cochlear symptoms. The repeated improvement from an injection can be very convincing to the patient and to the physician. Any

improvement occurring only after weeks and months of the same level of therapy, whether it be histamine or any other drug, is more likely due to the spontaneous remission from "nature's" tendency to re-establish autonomic balance than from a therapeutic result.

EVALUATION OF RESULTS OF TREATMENT.

It is difficult to evaluate the over-all results of treatment of Meniere's disease. No standard criteria have been established for such an evaluation. Too often the spontaneous remissions of the disease gain unjustified credit for the coincidental therapy. Furthermore, we must depend so frequently upon the subjective evaluation of the variation in symptoms by a wishful and anxious patient. The various hearing tests with tuning forks, audiometry and the more specialized tests for recruitment and discrimination, provide the best objective evidence for evaluation of cochlear changes; however, unless one has such records of a patient's hearing prior to the development of labyrinthine hydrops, there is uncertainty as to whether treatment has completely reversed the reversible cochlear pathology.

The above mentioned difficulties in evaluation became particularly apparent when I attempted to appraise the treatment received by 55 new cases of Meniere's disease in the common otologic practice of Dr. George E. Shambaugh, Jr., and myself from July 1, 1952, to July 1, 1953. For that year 362 new cases of perceptive deafness were reviewed, selecting 55 cases of unquestionable Meniere's disease. Of these 42 had reasonably definitive results to evaluate, and 13 were inconclusive, due either to an inadequate trial of therapy or to inadequate records. The changes effected in the principal cochlear and vestibular systoms were classfiied as completely relieved, moderately improved, slightly improved, and not relieved at all. Complete relief implied disappearance of any vertigo, and the previously fluctuating hearing loss stabilized at a normal level and absence of tinnitus and aural pressure. Moderate improvement meant much less frequent and severe attacks of vertigo, and the hearing stabilized at a significantly improved level, while tinnitus and sense of pressure have become minimal and possibly intermittent. Slight or no relief in the various symptoms hardly needs explanation.

Thus the varied relief of the individual symptoms is as follows:

VERTIGO: Of the 42 cases, 34 had vertigo; 18 showed complete relief, and five were moderately improved, totalling 23 or 68 per cent.

IMPAIRED HEARING: A significant loss in the speech frequencies of 15 decibels or over was present in 35 cases, of which only six showed complete relief and nine were moderately improved, totalling 15 or 43 per cent.

TINNITUS: Of 34 patients with tinnitus, nine had complete relief and nine were moderately relieved, a total of 18 or 53 per cent.

FULLNESS OF PRESSURE: This was the least common symptom, occurring in 28 patients of the 42. Of these 12 had complete relief and six had moderate relief, a total of 18 or 64 per cent (see Table I).

An interesting comparison between the above percentages of relief can be drawn with a report by Shambaugh[12] on histamine treatment of a labyrinthine hydrops-like syndrome in fenestrated ears, months or years after surgery. The respective percentages of relief in a much longer series of 370 patients for the symptoms of vertigo, drop in hearing and tinnitus were 65, 35 and 60 per cent.

Of the 42 cases, 25 had the complete triad of symptoms of Meniere's disease.

Six cases were classified as allergic in etiology, and in each one specific allergic management resulted in moderate or complete relief of all symptoms.

Only one case in this group of 55 cases underwent a labyrinthotomy because of incapacitating vertigo. After six months of complete relief, this patient has developed tinnitus, pressure and impaired hearing in the opposite ear, which is responding only partially to dilute histamine.

TABLE I.

RESULTS OF NON-SURGICAL MANAGEMENT OF MENIERE'S DISEASE IN 42 PATIENTS—BY SYMPTOMS.

Vertigo	34 Cases	68 Per Cent	Relieved or Improved.
Tinnitus	34 Cases	53 Per Cent	Relieved or Improved.
Hearing Loss	35 Cases	43 Per Cent	Relieved or Improved.
Aural Pressure	28 Cases	64 Per Cent	Relieved or Improved.

CASE REPORTS.

The audiograms in the case reports are shown without bone conduction recordings; however, these tests along with the tuning fork results of positive Rinne's and shortened Schwabachs in the involved ears all were consistent with the diagnosis of a perceptive deafness. Although the discrimination scores are recorded only in the first case report, all these patients gave descriptions of their hearing being "out of tune," "distorted" or "fuzzy." Since most otologists still do not do routine discrimination tests in their office practices, reliance upon such subjective comments as an indication of impaired discrimination is justifiable.

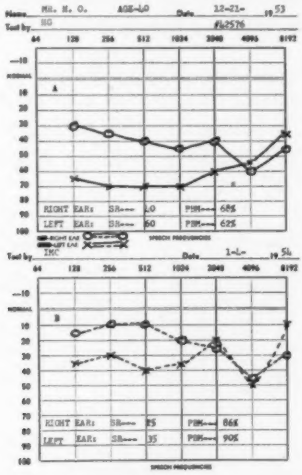

Fig. 2.

Case 1.—H. O., a man aged 40, was referred to me by his psychiatrist who desired an opinion as to whether a bilateral hearing loss of three months' duration was psychogenic or organic. A careful history defined the hearing loss as gradual in onset and progression rather than fluctuating in character. No vertigo or tinnitus were noted; however, the patient, an excellent pianist, volunteered that he noted considerable difficulty in pitch perception, as though he were "out of tune."

Examination revealed normal ear, nose and throat findings. The tuning fork findings confirmed the air and bone audiometry (see A in Fig. 2) which revealed a bilateral relatively flat perceptive loss, greater in the left ear. Diplacusis was demonstrated with the 256, 512, 1024 and 2048 tuning forks heard as higher pitched in the left ear. The discrimination scores, using the P B Max test, were quite low in both ears (see A in Fig. 2).

Except for a possible emotional factor, no obvious etiology was evident. The salt-free diet, potassium chloride and histamine management were started. The patient reported that "the right ear opened up" within an hour after receiving 0.1 cc. 1:100,000,000 histamine subcutaneously, and on the following day the "left ear opened up." Sublingual histamine, two drops twice daily, and a twice weekly histamine injection maintained the improvement in hearing, and the ears were no longer out of tune. This improvement is reflected in both the pure tone audiogram and the discrimination scores (see B in Fig. 2).

Comment: This case shows that treatment for a very possible etiologic emotional factor in cochlear hydrops by the psychiatrist did not obviate a trial of dilute histamine therapy, which we may conclude gave a positive and immediate response. Naturally, I realize that the case is too recent to evaluate as to whether it will be a maintained successful result; however, such a quick response to histamine, in my experience, augurs well for the eventual control of symptoms.

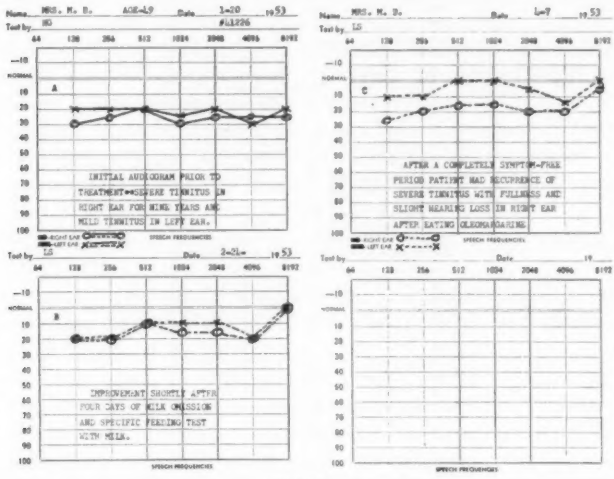

Fig. 3.

Case 2.—M. B., a married woman aged 49, was first seen on January 20, 1953, complaining bitterly of a constant tinnitus "like an African jungle or a jet plane" in the right ear for the previous nine years. This severe tinnitus often interfered with sleep and completely overshadowed the other symptoms of a fullness in the right ear, a recent mild tinnitus in the left ear, and a "possible" hearing loss (see A in Fig. 3). No vertigo has been experienced. Previous treatment had been surgery "for scar tissue in the back of the nose and throat," inflations and sedation.

The examination revealed normal drum membranes and middle ears. Audiogram (see A in Fig. 3) revealed a mild bilateral perceptive loss

with positive 256 Rinne's. No diplacusis was determined. Additional history was obtained of a constant, non-seasonal post-nasal discharge, but sinus X-rays were normal. There were numerous food and drug sensitivities elicited. Pork caused severe headache. Oranges, tomatoes and chocolate caused hives. Penicillin caused hives. Was once given a histamine (dosage ?) injection which caused fainting, hives and a watery nasal and eye drainage. As a child on the farm, the patient drank large amounts of milk, and now has indigestion from milk, so uses small amounts of milk, cream and butter.

In spite of a negative intradermal skin test to milk, the patient began a complete milk omission diet on February 18, 1953, and did a specific milk feeding test on the fifth day. Within ten minutes after the feeding of milk the patient developed sneezing, watery nasal drainage, coughing, nausea, cramps, a full feeling in the head and an increased "pounding" tinnitus. The audiogram three days later showed improvement in hearing (see B in Fig 3).

Complete milk omission has given complete relief from the tinnitus, and normal hearing, except for one occasion when patient ate oleomargarine which contains milk, and all symptoms recurred (see C in Fig. 3).

Comment: Two doses of dilute histamine were tried before the milk feeding test with mild reduction in severity of tinnitus lasting about two days after the injection; however, milk omission resulted in such complete relief that no further histamine was used. At the present time the patient still finds that she cannot tolerate even small amounts of milk.

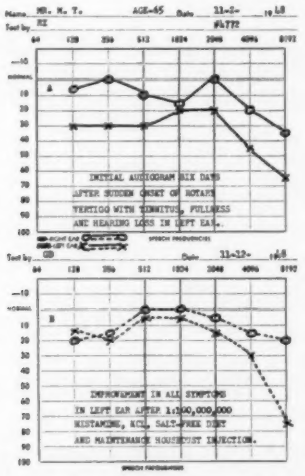

Fig. 4.

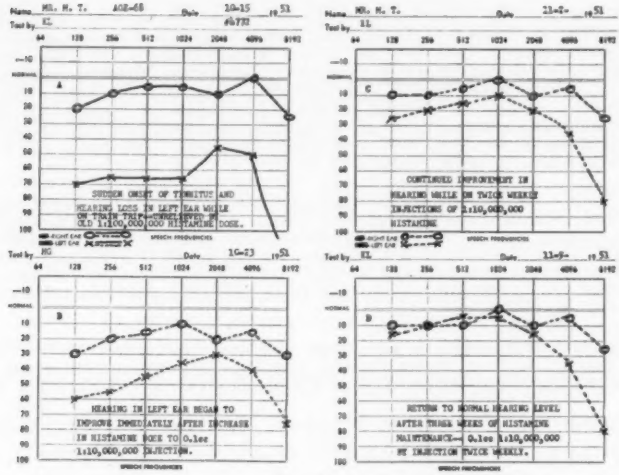

Fig. 5.

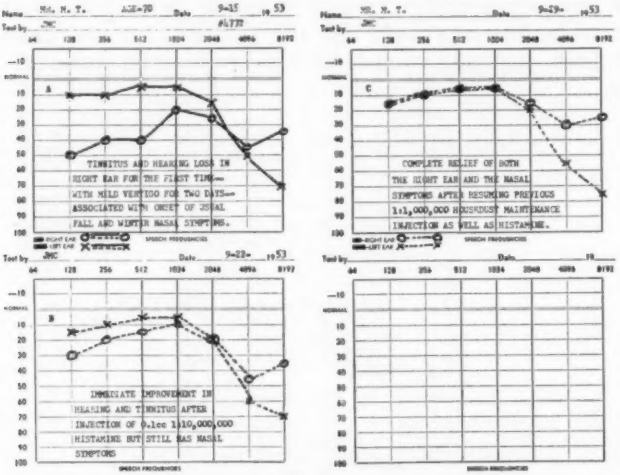

Fig. 6.

Case 3.—M. T., a man aged 70, has been a patient of the Shambaughs, Sr., and Jr., since 1915 because of a chronic yearly winter time recurrence of nasal blocking, nasal and post-nasal drainage, frontal and maxillary pain. On many occasions upper respiratory infections resulted in maxillary sinusitis necessitating irrigation. Following the realization of the importance of a sensitivity to house dust and the giving of house dust injections, the sinuses have not needed irrigation.

On November 2, 1948, the patient developed his first attack of tinnitus and impaired hearing in the left ear, followed the next day by a two-hour attack of rotary vertigo with nausea. The audiogram (see A in Fig. 4) and the tuning forks showed a perceptive deafness in the left ear particularly. In addition to his usual house dust injection, 0.1 cc. of 1:100,000,000 histamine was given. After the attack of vertigo on the following day, the tinnitus and hearing loss receded with a return to near normal hearing (see B in Fig. 4).

The patient had no particular difficulty, and in subsequent winters got along without many dust injections; however, on October 15, 1951, while on a train trip he was awakened by an attack of buzzing tinnitus and a blocked sensation in the left ear (see A in Fig. 5). Histamine, 0.1 cc. of 1:100,000,000 dilution gave no relief of symptoms, but following an increase to 0.1 cc. 1:10,000,000 histamine three days later, an immediate improvement began and continued while taking the same histamine dosage twice weekly, as well as two drops sublingually of 1:1,000,000 histamine. In addition a salt-free diet with potassium chloride was followed, and the patient's usual daily thyroid intake was increased from 2½ gr. to 3 gr. The progressive improvement can be followed in subsequent audiograms (see B, C and D in Fig. 5).

The next recurrence of Meniere's disease occurred about two years later in September, 1953, coincidental with the development of his usual autumn and winter nasal symptoms. This time the right ear was involved (see A in Fig. 6). The same histamine injection as in the last previous attack was given (1:10,000,000) and immediate improvement in the right ear occurred (see B in Fig. 6), but nasal symptoms persisted. The addition of 0.1 cc. of 1:1,000,000 house dust to the histamine injection was followed by complete clearing of the right ear and nasal symptoms (see C in Fig. 6).

Comment: Whether the long-standing house dust nasal allergy had any causal relationship to the Meniere's disease is not so easy to prove as in the previous food sensitive patient; however, it is interesting that the hydrops attacks all occurred in the fall, coincidental with or following the usual exacerbation in a chronic house dust allergy at that time of the year.

SUMMARY AND CONCLUSIONS.

An attempt has been made to evaluate the results of the small optimum dosage of histamine in the treatment of Meniere's disease by studying 55 new cases seen in one year.

Standard criteria by which to judge results in the medical management of Meniere's disease are sorely needed.

As a rule, if histamine is going to be effective, the relief from symptoms is prompt and definite within hours after injection.

Although only six cases of the 55 (11 per cent) could be shown to be of specific antigen-antibody allergic origin, this

possible cause should not be overlooked. In our experience, the specific allergic cases properly managed give the best possible results.

REFERENCES.

1. HILGER, J. A.: Autonomic Dysfunction in the Inner Ear. THE LARYNGOSCOPE, 59:1-11, 1949.

2. HILGER, J. A., and GOLTZ, N. F.: Some Aspects of Inner Ear Therapy. THE LARYNGOSCOPE 61:695-717, 1951.

3. WILLIAMS, H. L.: Allergy of the Inner Ear. *Arch, Otolaryngol.*, 56:24-44, 1952.

4. WILLIAMS, H. L.: Meniere's Disease, Ed. 1, Springfield, Charles C. Thomas, 1952.

5. WRIGHT, A. J.: Meniere's Disease. *Proc. Roy. Soc. Med.*, 41:801-805, 1948.

6. SELFRIDGE, GRANT: A Survey of the Relation Between Nutrition and the Ear. *Ann. Otol. Rhinol. and Laryngol.*, 49:674-687, 1940.

7. GUNDRUM, L. K.: Etiologic Analysis of One Hundred Cases of Meniere's Symptom-Complex. *Arch. Otolaryngol.*, 57:123-128, 1953.

8. MYGIND, S. H., and DEDERDING, DIDA: The Pathogenesis of Meniere's Disease and of Kindred Conditions in the Ear and the Rest of the Body. *Ann. Otol., Rhinol., and Laryngol.*, 47:938-946, 1938.

9. FURSTENBERG, A. C.; LASHMET, F. H., and LATHROP, FRANK: Meniere's Symptom-Complex: Medical Treatment. *Ann. Otol., Rhinol., and Laryngol.*, 43:1035-1046, 1934.

10. RINKEL, H. J.: Food Allergy. II. The Technique and Clinical Application of Individual Food Tests. *Ann. Allergy*, 2:504-514, 1944.

11. PERLMAN, H. B.; GOLDINGER, J. M., and COLES, J. O.: Electrolyte Studies in Meniere's Disease. THE LARYNGOSCOPE, 63:640-651, 1953.

12. SHAMBAUGH, G. E., JR.: Histamine in Treatment of Certain Types of Headaches and Vertigo Following the Fenestration Opeartion. *Arch. Otolaryngol.*, 51:781-785, 1950.

DEUTSCHE GESELLSCHAFT DER HALS-NASEN-OHRENARZTE.

The 25th Annual Meeting of the Deutche Gesellschaft der Hals-Nasen-Ohrenarzte will be held June 2 and 5, 1954, at Marburg-Lahn. For further information, address Prof N. Mittermeier, Marburg-Lahn, Germany.

SALIVARY GLAND TYPE TUMORS OF THE HEAD AND NECK.*†

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Salivary gland-type tumors occurring in aberrant locations about the head and neck have been of interest ever since Robin[9] described a "mixed tumor" of the palate in 1852, and Forster[3] recognized a similar nasal tumor in 1859. Despite the fact that recently multicentric tumors of this variety were produced in animals by inoculation with a filterable virus, their etiology has never been satisfactorily explained, and in each theory concerning their nature it is hard to explain all of the different constituents. They are now generally considered to be purely epithelial in origin, although McFarland[5] and some other pathologists do not hold this view. If we accept the epithelial origin, the original term of "mixed" tumor by which they are commonly designated must be discarded. No one type of treatment has found universal acceptance, and in the clinical discussions a wide variation in the type of treatment was noted. End result studies of treatment are infrequent, for the exact assessment of the frequency of recurrence and subsequent outcome is difficult, because of the variable and often long intervals before recurrence.

It has been estimated by Willis[11] that 10 per cent of these tumors occur in locations other than the salivary glands, with approximately half in the palate. In this report of 19 aberrant mixed salivary gland-type tumors, the youngest was 13 years and the oldest 68 years, with an average age of 46. There were nine males and ten females. Six were situated in the palate, two in the nasal septum, two in the sinuses, seven in the pharynx, and one each in the larynx and trachea. While

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nine of the cases exhibited recurrence or extension only two metastasized, and these were to the cervical lymph nodes. Five patients have died, and one is living with tumor (see Table 1).

TABLE I.
Salivary Gland-Type Tumors in Aberrant Locations.

Location	No.	Treatment		Recurrence	Living	Dead
		Surg.	Surg. Irrad.			
Septum	2	1	1	2	1	1
Sinuses	2	1	1	2	1	1
Palate	6	6	0	0	6	0
Pharynx	7	7	0	3	4	3
Larynx	1	1	0	1	1	0
Trachea	1	0	1	1	1	0
Total	19	16	3	9	14	5

Pleomorphism is a prominent feature, and any or all varieties of structure may be found in one tumor. They may resemble simple adenomas on the one hand, or frankly malignant anaplastic carcinomas on the other. On the whole, the growths outside of the salivary glands seem to have a higher degree of malignancy than those in the salivary glands themselves.

Most salivary gland-type tumors occupy a borderline position. They are often classed as benign, but since they have been demonstrated microscopically to be nonencapsulated, recur after removal, widely infiltrate the surrounding tissue, and occasionally metastasize, this designation is not tenable. Likewise, it does not seem accurate to classify them as benign tumors which subsequently become malignant, although Slaughter, Southwick and Walter[10] found 12 out of 212 cases in the parotid gland itself which were thought to have originated from pre-existing benign tumors. They should be considered as malignant from the beginning, although their rate of proliferation and degree of malignancy may sometimes show more or less abrupt increases. The most reasonable concept is to regard them all as low grade malignant adenocarcinomas, regardless of the histologic detail. The question to be answered is not whether the tumor is benign or malignant, but how innocent or malignant is the particular tumor?

Encapsulation of salivary gland-type tumors occurring in

aberrant locations is seldom complete. Even when grossly the margin of the tumor appears sharply delimited and encapsulated, microscopic examination will commonly reveal extension into the surrounding area. Because of this the risk of postoperative recurrence is considerable when a sharp line is hewn between the visible tumor and the adjacent tissue. Although the origin of these tumors from multiple foci, with

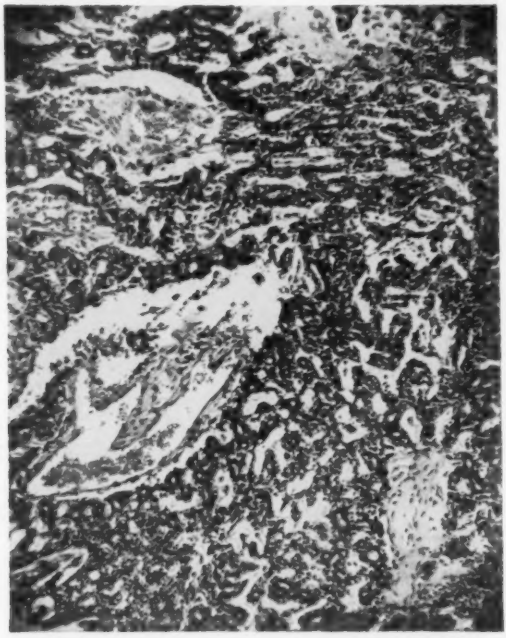

Fig. 1. Histologic picture of salivary gland type tumor of nose. The closely packed masses of cells with apparent invasive tendencies was considered malignant, yet the patient remained well for eight years after local excision. Recurrence was treated by excision with a four-year follow-up free from disease.

subsequent development of a new tumor, must be considered in recurrent cases, the usual cause for recurrence is conservative or incomplete surgical removal.

At present an accurate prediction regarding the outcome of any one tumor cannot be made, and it is questionable whether any salivary gland-type tumor can ever be regarded

as completely cured. Certainly the microscopic structure of a salivary tumor is an unreliable guide to prognosis. Recurrence may follow removal of tumors of the most benign-looking histologic type, while cellular active-looking growths, from which recurrence and local invasion was anticipated, may fail to show malignant tendencies. In the following case with involvement of the nasal septum, histologic study revealed masses of cells which appeared to be rapidly growing without limitation by fibrous tissue or a capsule, leading to an initial diagnosis of basal cell carcinoma (see Fig. 1), yet eight years elapsed after local removal of the tumor before a recurrence developed.

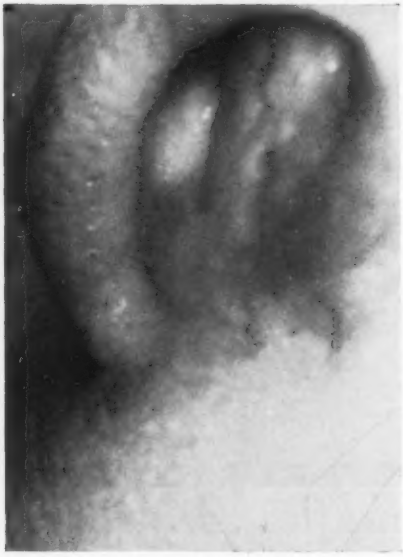

Fig. 2. Firm, discrete, slightly nodular salivary type tumor of cartilaginous and bony septum projecting into right nasal chamber.

Case 1. A 48-year-old male first noticed difficulty in breathing through the right side of the nose in 1934. For the next 10 years polyps were removed from both sides of the nose at frequent intervals. Both the Wassermann and Kahn reactions were strongly positive. In 1944 the blockage on the right became almost complete, and he was admitted to Jefferson Hospital, where a mixed salivary gland-type tumor of the nasal septum was found (see Fig. 2). Surgical excision of the tumor and involved portion of the septum was done.

The patient remained well until 1952, at which time he returned with recurrence of the tumor in the floor of the nose and posterior septum. There was no involvement of the turbinates or sinuses. At operation the remaining septum and the mucosa of the entire floor of the nose was removed through an alveolar approach, and an area of bone destruction in the hard palate approximately ½ cm. in diameter was uncovered. The buccal mucosa was intact over this area, and curettage of the edges of the exposed bone was done without additional bone removal. It would seem likely that the tumor originated in the palate, and extended upward into the nasal septum.

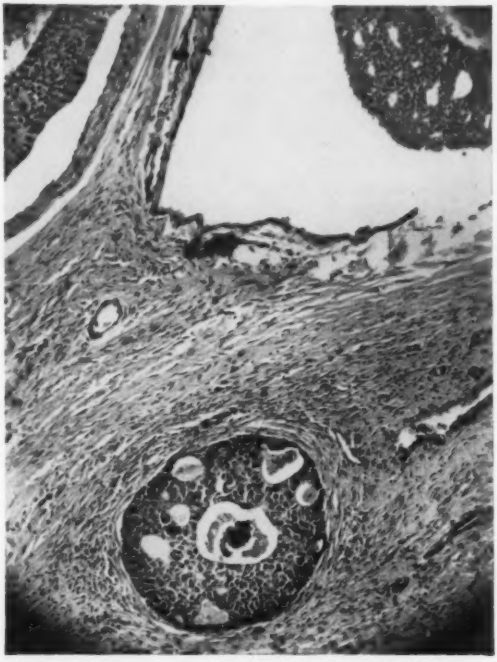

Fig. 3. Histologic section of salivary gland type tumor of antrum. Because of the discrete acini surrounded by large areas of fibrous tissue, with distinct encapsulation the lesion was diagnosed as benign. Despite radical surgical removal the patient died one year later from intracranial extension.

To illustrate further the unreliableness of the histologic structure in prognosis and treatment, the histologic appearance of sections taken from a tumor which involved the antrum and lateral nasal wall revealed discrete acini with a large amount of fibrosis, which was thought to be relatively benign and non-invasive (see Fig. 3). This patient died

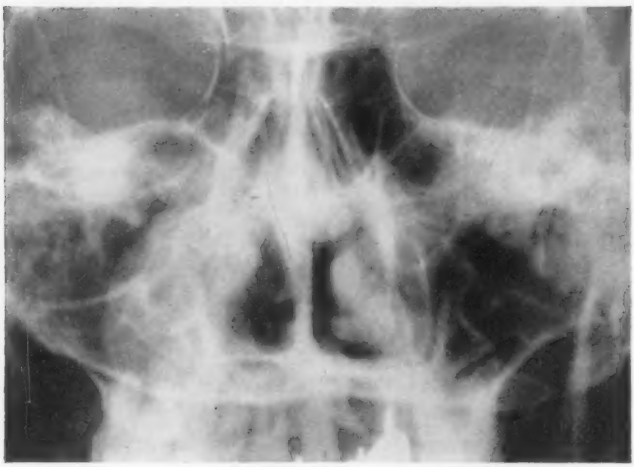

Fig. 4. Roentgenogram with a large, smooth, circumscribed tumor of the medial wall of the right antrum, which proved to be a salivary gland-type tumor.

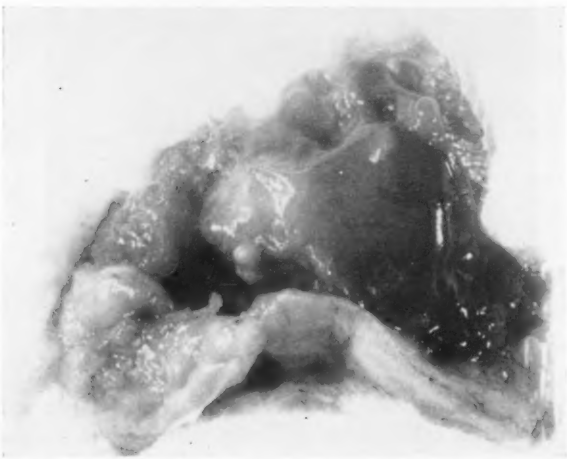

Fig. 5. Specimen of large encapsulated antral salivary tumor with hard palate and excised normal tissue around the periphery of the lesion (inner aspect).

within a year from local extension of the tumor into adjacent intracranial structures.

Case 2. Difficulty in breathing through the right side of the nose had been noticed for two months by a 49-year-old white female. For a few weeks fullness and tightness along the right side of the nose and occasional blood tinged secretion from that side had been present. She had also observed slight swelling over the right cheek.

Examination revealed soft tissue swelling of the right cheek, and narrowing of the right nasal airway due to protrusion medially of the lateral wall. On the roentgenogram (see Fig. 4) a density in the floor and medial wall of the right maxillary sinus was observed. Tissue was removed intranasally, and reported as a salivary gland tumor, benign type.

At operation through an external approach the tumor was found to extend up to the ethmoids and down to the hard palate. The tumor mass was removed as a block, which included the hard and soft palates, lateral nasal wall, and antral floor (see Fig. 5). Thorough electrocoagulation of the remaining tissue was performed. The patient was fitted with a prosthesis and discharged from the hospital.

Eight months later diplopia developed with recurrence of the tumor in the right ethmoid area. There was paralysis of the right Vth and VIth cranial nerves, and roentgenograms revealed extension of the destructive process into the sphenoid and base of the skull. Irradiation therapy using a tumor dose of 4860r over 26 days was carried out with little improvement. Death occurred four months later.

The symptoms are largely due to obstruction. When the growths reach a large size, pressure symptoms or distortion develops. Pain is almost invariably absent. Although in the nasal tumors a feeling of fullness and tightness about the head was described, obstruction to breathing on the involved side, together with some secretion which at times was blood tinged, was the major complaint. In the palatal cases the patients complained of difficulty with chewing and swallowing and frequently noticed swelling, but there was no ulceration or bleeding. Difficulty in swallowing and eating was prominent when the pharynx was the site of the lesion. Hoarseness was the main symptom in the laryngeal case, and obstruction to breathing, especially when lying down, occurred in the tracheal case.

The palatal tumors were not found in the midline but near it on one or the other side. Since there are no glands in the anterior part of the hard palate, the common location was in the posterior portion of the hard palate or in the soft palate. Although excavation and perforation of the palatal bone was present in three of the palatal cases it did not denote invasion. When the tumor was fixed to the underlying bone the resulting crater was due to pressure atrophy. These tumors

were rounded, well circumscribed, not ulcerated, and could easily be separated from the surrounding tissue. No recurrences have been observed as yet in this group. An illustrative case of a palate tumor follows:

Case 3. A 56-year-old female had a "growth in the throat" for 32 years. The year previous to seeking medical advice and treatment she had observed a gradual increase in the size of the tumor, for which she received "needles" and vitamin therapy from her local physician to shrink the lump.

On anterior rhinoscopy a large tumor mass was visualized on the right side of the nasopharynx arising from the soft palate. When the oral cavity was examined a large, firm, smooth, rounded mass occupying the right half of the soft palate and extending across the midline to the opposite side was noted (see Fig. 6).

When the mass was removed it was found to be limited to the soft palate and faucial region. The pathologic report was mixed tumor.

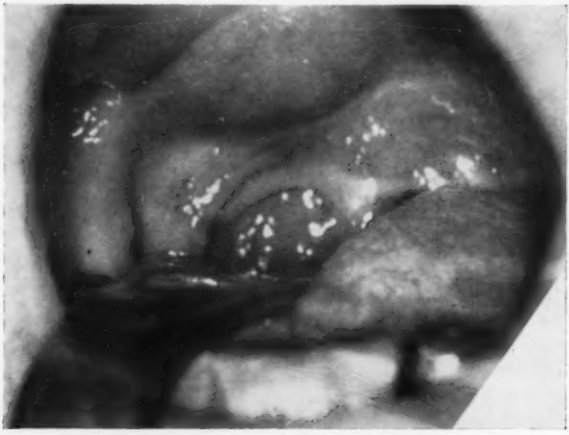

Fig. 6. Appearance of salivary gland tumor of palate. The lesion at the junction of the hard and soft palate on the right side can be seen as a smooth, round mass projecting behind the denture.

In the four cases involving the nose and the sinuses, all developed recurrences. One patient with involvement of the maxillary and ethmoid sinuses died less than one year after the original diagnosis and treatment. Death in this case was due to direct extension of the tumor into the cranial structures, but there was no evidence of regional or distant metastasis. In the other case of a reported benign lesion in the antrum, recurrence occurred after an extensive local removal, and resection of the upper jaw was then required. Only

after histologic examination of the entire maxilla was our suspicion confirmed and the diagnosis of a malignant tumor made.

In the seven pharyngeal cases there were three recurrences and three deaths. One of the patients that died developed cervical metastasis. The following patient is living after five years, even though the pathologist considered the tumor malignant and the non-encapsulated mass extended to the base of the skull, so that it was impossible to excise it in one piece, but removal had to be accomplished by morcellation.

Case 4. A 53-year-old white male complained of a sore throat with a feeling of "lump in the throat" for one month. On examination a prominence of the right side of the pharynx occupying the region of the faucial tonsil was observed. The mass was very firm and extended outward to the hard palate, laterally to the mandible and downward to the base of the tonsillar fossa.

At operation the tumor was found to be well encapsulated, extending upward to the base of the skull. As the dissection was performed the upward prolongation broke off and was removed by morcellation. It was not known whether all of the capsule had been removed, but subsequent study failed to reveal any further evidence of the tumor.

Although the pathologic report was malignant mixed tumor of the pharynx, he has had no symptoms and remained free from recurrence for six years.

In the laryngeal case local recurrence occurred in less than two years. Subsequently metastasis to the cervical lymph nodes developed, so that laryngectomy and block dissection of the lymph nodes of the involved side of the neck was necessary.

Case 5. Hoarseness and soreness in the throat of two months' duration were the chief complaints of a 55-year-old male. These symptoms followed a cold, and recently the patient had noticed a sticking sensation in the left side of the neck referred to the left ear.

On examination a marked widening of the vallecula on the left side with displacement of the upper part of the epiglottis posteriorly and crowding of the left aryepiglottic fold to the left, due to a mass, was observed.

Operation was performed through a lateral thyrotomy and the tumor which involved the left ventricular band and pre-epiglottic space was excised. The histologic diagnosis was salivary gland tumor of the larynx. Six months later a recurrence was noted in the left vallecula. He was operated upon through a transhyoid approach and excision of the mass, which involved the pre-epiglottic space extending to the base of the tongue and the lateral pharyngeal wall, was done. The tumor was encapsulated and separated readily from the surrounding tissue. Two years after the second operation the patient returned complaining of blood spitting, and an ulcerating recurrent lesion was found on the posterior surface of the epiglottis. In addition there was an indurated tender area in the left submaxillary region. This time a total laryngectomy with left radical neck dissection and excision of the right submaxillary gland was performed. Histologic examination revealed adenoid

cystic carcinoma of the larynx with metastasis to lymph nodes and submaxillary gland.

In the case of the tracheal tumor wide infiltration was observed at the time of operation, so that the entire tumor could not be removed. The patient is living with the tumor, but just how long she will survive before succumbing to the disease is problematical.

Case 6. A 52-year-old female complained of difficulty in breathing, more marked on exertion, and a huskiness of the voice and wheezing at times, of one year's duration. A loss of 17 pounds of weight had occurred.

On indirect and direct laryngoscopic examination there was found a paralysis of the left vocal cord and a large, red mass on the anterior tracheal wall approximately 1½ to 2 cm. below the cord level. The mass almost completely filled the tracheal lumen, leaving an airway of only a few millimeters in the posterior portion. A portion of the tumor was removed, and the histologic diagnoses was mixed tumor of the salivary gland type.

After a tracheotomy had been done the neck was surgically explored, and the tumor was found to involve the larynx and upper trachea with extensive infiltration into the surrounding tissues. The patient received irradiation therapy in the amount of 3500r. to both sides of the neck, with little improvement in the tracheal narrowing or the extent of the tumor. Radical surgical excision is being considered, but as yet has not been done.

Treatment of aberrant salivary gland-type tumors must always be individualized to a certain extent, but the institution of definite therapy as early as possible in every case is fundamental.

To follow pathologic interpretations alone may result in unnecessarily extensive operations, but what is more hazardous they may lead to conservative and incomplete procedures when extensive surgery is needed. The selection of treatment must be based on the coordinated clinical and pathological findings, for the location of the tumor, as well as its histologic structure is a contributing element in determining the extent of therapy. With the possible exception of the palatal tumors the only factor that seems to have any real influence on the final outcome is the radicality of the initial surgery. We can not agree with Dean,[2] who contended that mutilating operations should not be done when dealing with these semi-malignant tumors.

Although the results of surgical treatment have not been encouraging, we believe it remains the best method of treatment. Because these tumors usually have an incomplete capsule, enucleation alone cannot be relied upon as the chief

surgical procedure. Adequate tissue removal around the periphery of the lesion, including both the soft and necessary bony structures, is the most effective means of preventing recurrence. When the extensive surgery always needed to extirpate these tumors in aberrant locations other than the palate is done initially without waiting for recurrence, the recurrence and mortality figures will improve. Cervical lymph node metastasis should be treated simultaneously with the primary tumor by radical neck dissection.

Since the situation of these tumors in the upper air and food passages, especially when they are located in the ethmoid region, may prevent the ideal surgical procedure of radical removal with the capsule and adjacent normal tissue, the operative procedure described by Hybbinette[4] deserves consideration. This surgical approach consists of incision of the capsule with removal of the tumor contents within the capsule. After this has been done the capsule is dissected free by sharp and blunt dissection and totally excised. This method has merit in the large and bulky tumors in this region, because it allows a careful removal of the capsule after the real tumor is out of the way and the working space is increased. One of our cases was treated in this manner because of the large size and extent of the tumor. This patient also received preoperative X-ray therapy and implantation of radium seeds, but developed recurrence within a year, and was dead within four years.

Certain of the European investigators, notably those in the Scandinavian countries, have a high regard for irradiation therapy. Ahlbom[1] thought the best results were obtained with irradiation, combined with surgical therapy. Pre- and postoperative irradiation was administered in those cases with manifest or suspected malignancy. In the clinically benign types no irradiation was given preoperatively but postoperative therapy was used. Ohngren[7] expressed a similar view, but advocated electrosurgical treatment. Ringertz[8] utilized both irradiation and electrosurgery, as described by Ohngren.

The case for the use of preoperative irradiation has been given as: *1.* the tumors are made technically easier to remove; *2.* prevention of recurrence, and *3.* the clinical diagnosis may

be mistaken, and the inflammatory lesion will subside under irradiation therapy. It is difficult for us to justfy this procedure, for these tumors have proved so highly resistant to radiation that no real effect has been secured. Of secondary importance is the subsequent fibrosis with which the surgeon has to deal at operation. The high degree of radio-resistance of this group of tumors likewise militates against the success of postoperative irradiation, although on occasion irradiation has seemed beneficial. It is doubtful if its routine use postoperatively has an effect on the prevention of recurrency.

Because of the relative slow growth of this type of tumor and its late metastasis, the mortality should be very low. To date our figures together with those of others, New and Childrey,[6] Ohngren,[7] and Ringertz,[8] have not borne out this optimism (see Table 2). It is questionable whether previous operative trauma has a direct bearing upon recurrence. Failure to appreciate the need for radical surgical procedures would seem to be responsible for the high mortality rate.

TABLE II.

Recurrences and Mortality in Reported Series of Cases.

Author	No. of Cases	Recurrences	Deaths
New and Childrey	48	28	11
Ohngren	11	3	3
Ringertz	37	15	14
Putney and McStravog	19	9	5
Total	115	55	33

SUMMARY.

The slow growth and clinical encapsulation of many of the aberrant salivary gland-type tumors makes us inclined to overlook their potentialities, and influences our surgical judgment. Except for some of the palatal lesions these tumors about the head and neck possess the characteristics of low grade malignancies. Removal of the entire lesion, together with an adequate margin of normal soft and bony tissue utilizing surgical procedures, is preferred over other forms of treatment, but this may not be sufficient to prevent recurrence. In our experience, the addition of irradiation therapy has

proved no more beneficial than surgery alone. In a group of 19 cases, six were located in the palate and have remained free from disease. Of the remaining 13 cases five have died, two had metastasis and nine developed recurrence.

REFERENCES.

1. AHLBOM, H. E.: Mucous and Salivary Gland Tumors. *Acta. Radiologica.* Suppl. 23:1, 1935.

2. DEAN, L. W.: In Discussion on New, G. B., Mixed Tumors of the Throat, Mouth and Face. *J.A.M.A.*, 75:732, Sept. 11, 1920.

3. FORSTER, A.: Schlumcancroid, Etc. *Atlas d mikr. path. Anat.*, 1859.

4. HYBBINETTE, S.: Contributions a l'Stude des Tumeurs Benignes de la Parotide et a leur Traitement Radio Chirurgical. *Acta. Chir. Scan.*, 77:19, 1935.

5. MCFARLAND, J.: The Histopathologic Prognosis of Salivary Gland Mixed Tumors. *Amer. Jour. Med. Sci.*, 203:502, 1942.

6. NEW, G. B., and CHILDREY, J. H.: Tumors of the Tonsil and Pharynx. *Arch. Otolaryng.*, 14:699, Dec., 1931.

7. OHNGREN, L. G.: Malignant Tumors of the Maxillo-ethmoidal Region. *Acta. Otolaryngol.* Suppl. 19, 1933.

8. RINGERTZ, N.: Pathology of Malignant Tumors Arising in the Nasal, Paranasal Cavities and Maxilla. *Acta. Otolaryngol.* Suppl. 27:52, 1938.

9. ROBIN, E. P.: Note sur Quelques Hypertrophies Glandulaires. *Gaz. d. hop. Paris*, 1852, XXV.

10. SLAUGHTER, D. P.; SOUTHWICK, H. W., and WALTER, L.: Fate of Recurrent or Persistent Parotid Tumors. *Surg., Gynec., and Obstet.*, 96: 535, May, 1953.

11. WILLIS, R. A.: Pathology of Tumors, p. 320, 1948. The Mosby Co., St. Louis.

OBSERVATIONS ON FRONTAL SINUS DISEASE REQUIRING EXTERNAL SURGERY SINCE 1945.*

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The observations which I am presenting are based upon the clinical and pathological study of 20 cases of frontal sinus disease, which were operated upon externally since 1945. The purpose is to illustrate the clinical manifestations of disease in this sinus during the era of chemo-therapy and bio-therapy, and to present an appraisal of its management.

I have divided the cases into two groups on the basis of nasal symptoms and rhinoscopic findings because these clinical manifestations are commonly considered characteristic of sinus disease. On this basis it was noted that such symptomatic evidence occurred in only six patients, while it was absent in 14. My interest was principally directed to the 14 patients with absent nasal symptoms because they illustrated a wide variety of lesions with diverse clinical manifestations, none of which were referred to the nose.

I believe it is important to emphasize this because too frequently diagnosis and therapy are based on the idea that sinus disease implies nasal symptoms and nasal symptoms imply sinus disease, so I will comment on these patients first. Their many interesting clinical and pathological features warrant a detailed discussion, but as there is not enough time for this I will group them according to similar outstanding clinical features, and will use short extracts from the clinical records and Roentgen findings of one case, to illustrate each group.

The 14 cases can be separated into two main groups, consisting of 10 in which obstruction, partial or complete, of the sinus outlet to the nasal cavity was the important feature, and four in which the primary pathology was outside the sinus and the sinus was secondarily involved. In the 10 cases of obstruction of the sinus outlet the clinical features differed

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because the pathological reaction in the osseus walls was variable. This reaction was one of atrophy due to pressure from an expanding fluid in some, and one of hyperplasia and thickening from inflammation in others. In three of them an external fistula was also present, which entered the sinus through a perforation in its floor.

Six of the 10 cases showed Roentgen findings characteristic of a mucocele, such as absence of scalloping of the sinus wall, increased translucency, and flattening of the medial superior margin of the orbit. In three of these mucocele-like lesions there was an accumulation of a fluid mass in the sinus, with distention of all its walls and marked atrophy of mucous membrane and bone, but there was no sign of any inflammatory reaction or residue. The location of the obstruction varied: in one, it was at a distance from the sinus outlet in the ethmoid capsule, in the other two, it was at the sinus outlet. The cause of the obstruction was obscure in all three cases. A typical example of this lesion is:

J. B., male, age 48, complained of right frontal headache and ptosis of the right eye of three months' duration and gave no history of any

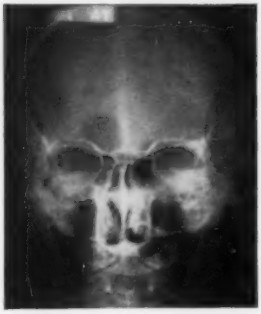

Fig. 1. Roentgenogram of right frontal sinus showing smooth contour, increased translucency, absence of normal densities of sinus walls the result of pressure atrophy of expanding fluid mass retained by blockade of sinus outlet. No inflammatory reaction.

sinus infection. He had suffered from a linear fracture through the right parietal and temporal bone at the age of 12 due to a fall, which had caused marked deafness in the right ear, but no connection could be established between it and the present condition. The rhinoscopic examination was entirely negative. He had been seen by an ophthalmologist, fitted with glasses, and referred to a neurologist before an investigation of the sinuses was sought.

Roentgen examination of the right frontal (see Fig. 1) showed typical findings of a mucocele; smooth contour, increased translucency, absence

of normal densities of the sinus wall. Iodized oil was instilled into the sinus by pushing a needle through the anterior wall, which was as thin as eggshell, and it demonstrated complete blockings of the naso-frontal duct in the ethmoid region. Cure was obtained by simply removing some of the anterior ethmoid cells and relieving the blockade into the nose. The sinus contained heavy grayish mucoid liquid, but no signs of any inflammatory reaction.

Trauma caused a sinus lesion in three patients who showed Roentgen findings of a mucocele without nasal symptoms or rhinoscopic evidence of disease. In them, however, a marked inflammatory reaction was present, with swollen hyperplastic mucous membrane, fibrotic granulation tissue and thickening of the bony walls, instead of atrophy (see Fig. 2). In one of these patients a calcareous mass, due to calcification of a hematoma, had formed in the sinus wall which partly invaded the orbit. There was no evidence of infection in any of them, and the sinus blockade, which caused the mucocele syndrome, was entirely due to inflammatory residue, the result of trauma.

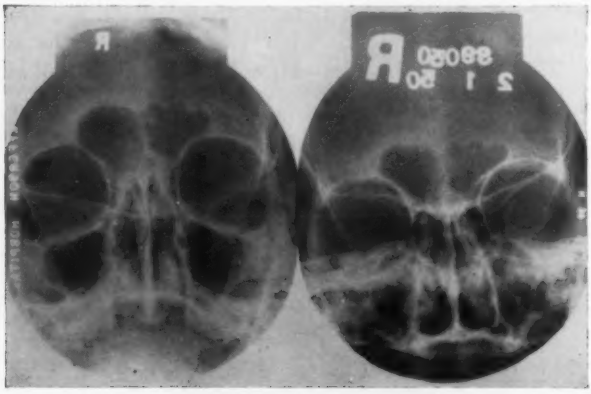

Fig. 2. Roentgenogram of right frontal sinus showing features similar to those in Fig. 1.—there is absence of scalloping, increased translucency, flattening of the medial orbital margin, and in addition increased perisinus density from sclerosis, caused by direct trauma from striking forehead against a door.

Cure was obtained in all three by simply removing the obstructing inflammatory tissue and re-establishing an adequate naso-frontal connection; however, the lesion existed in each case three months to one year before this treatment was carried out. An example is:

A woman, age 59 years, who complained of a tender swelling over the medial aspect of the right orbit and forehead, which developed after striking her head against a door three months prior to admission. It was large enough to interfere with turning her eye medially and upward,

and was smooth and compressible like a cyst. The Roentgen findings resembled those of a mucocele and were very similar to the first case (see Fig. 2).

They showed absence of scalloping, increased translucency and flattening of the medial margin of the orbit, but in addition increased perisinus density from sclerosis. External opening of the cystic swelling revealed a brown, thick, pasty material, swollen mucous membrane, and fibrotic granulation tissue which completely blocked the ostium of the sinus. No evidence of infection was present, and culture was negative. The clinical manifestations and Roentgen findings were due to pressure from the expanding fluid mass contained within the sinus by the blockade of its outlet with inflammatory residue. This lesion was first diagnosed as a probable dermoid cyst and its origin from the frontal sinus was not suspected until it was incised and the sinus cavity entered.

Mucoceles are commonly reported as specific lesions or disease entities involving nasal sinuses; however, observations from these six patients indicate that they are simply a peculiar physical state of the sinus; namely, an expanding fluid mass confined within the cavity by an obstruction of its outlet, and are only one phase of a disease process, the etiology of which may be varied.

There were four patients with absent nasal symptoms and rhinoscopic findings, whose illnesses began with acute cellulitis over the left frontal sinus and eye. Three of them developed an external fistula at the middle third of the brow, which existed with intermittent drainage for periods of five months, 10 months and 18 months, respectively, before the underlying pathology in the frontal sinus was investigated, and during this time a wide variety of futile therapeutic measures were applied, including hot compresses, sulfas, Roentgen therapy and antibiotics. One of the patients was hospitalized as an emergency, because of a generalized convulsive seizure which was due to localized osteomyelitis of the posterior wall of the sinus and pachymeningitis of the underlying dura. Another was hospitalized because of opacity of the cornea and chronic conjunctivitis due to deformity of the upper lid from scar tissue. Cure was obtained in all by external surgery to remove necrotic tissue, inflammatory debris and establish an adequate naso-frontal connection.

The patient with corneal opacity due to deformity of the

upper lid is taken as an example (see Fig. 3). She was a 65-year-old woman who gave a history which made no reference to the nose or sinuses. Four years prior to admission a painless swelling occurred on the left upper lid, which very slowly increased in size for a period of two years, when it rapidly got larger, red and very painful. It was diagnosed as

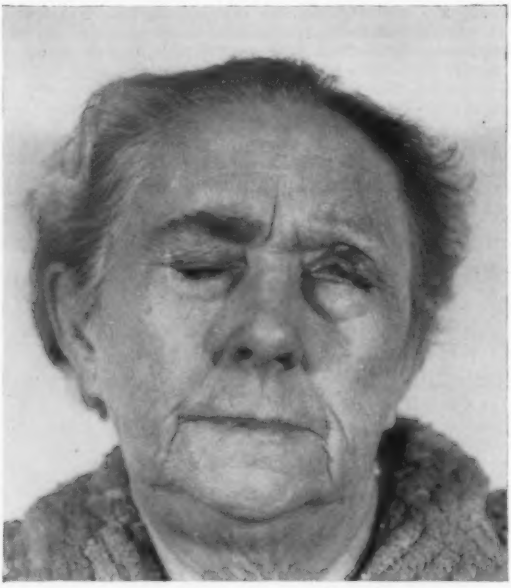

Fig. 3. Patient with fistula into the left frontal sinus of 10 months duration. Contracting scar tissue caused permanent retraction of upper lid with exposure of cornea resulting in opacity and blindness.

an abscess of the upper lid and was incised at the mid-point of the brow. Pus discharged, relieving the swelling, redness and pain. She was then treated over the following months with sulfas, penicillin and Roentgen therapy, but the discharging fistula persisted, and scar tissue gradually retracted the upper lid so that it was unable to cover the cornea, resulting in opacity, chronic conjunctivitis and blindness.

The Roentgen films (see Fig. 4), showed clouding of the left frontal sinus, thickening of its walls and marked con-

densing osteitis surrounding it. External operation revealed a defect in the floor of the sinus measuring 2 x .5 cm., the margin of which was atrophied to knife-like thinness. The sinus was a moderately large single cavity, with markedly thickened intact membrane except at the site of the fistula. The posterior and anterior walls were markedly thickened, and had a pebbled appearance. A probe was passed from the nasal chamber through a large normal anterior ethmoid cell, but was stopped by a thin partition of bone which sealed off the frontal sinus. When this partition was removed the ethmoid cell provided a very suitable naso-frontal duct.

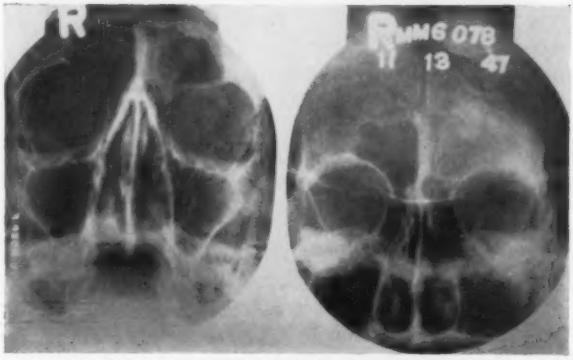

Fig. 4. Roentgenogram of the patient with fistula and corneal opacity, showing clouding of the left frontal sinus, thickening of its walls, and marked condensing osteitis surrounding it.

The biopsy report on the lining membrane of the sinus was pseudo-stratified, ciliated, columnar and transitional epithelium covering edematous vascular tissue which contained plasma cells and lymphocytes. Staphylococcus aureus was reported from the culture. The etiology of this lesion is obscure, and there is nothing in the history or clinical findings to account for the closure of the frontal sinus. Pressure from the contained fluid mass caused atrophy and finally perforation of the thin floor, but it did not expand the cavity where the walls were thickened by condensing osteitis.

These four patients again illustrate the futility of any treatment of closed lesions of the frontal sinus without establish-

ing an adequate naso-frontal connection. Two of them had active syphilis, which was probably a factor in the pathological development of the lesion, but it is interesting to note that although both received large doses of penicillin for treatment of their syphilis the antibiotic had no effect upon the frontal sinus disease, until nasal drainage was provided.

In four other patients with nasal symptoms and rhinoscopic findings so slight as to be of little diagnostic significance, the pathology causing the clinical manifestations proved at operation to be outside of the frontal sinus. In two of these the ethmoid sinuses were involved with an expanding lesion caused by obstruction to the cell outlet, which impinged upon the frontal sinus. The other two were instances of frontal bone cysts encroaching upon the frontal sinus; one an epidermoid, the other a cyst of traumatic origin. Frontal bone cysts are of interest to us because they illustrate that the frontal bone can be the site of cavities other than the frontal sinus, and they must be considered in any careful diagnostic study of frontal sinus disease.

I cite as an example a patient with a cystic lesion due to trauma, which was immediately adjacent to a chronically infected frontal sinus. She was a woman aged 67 years, with a history of prolonged nasal treatment and right middle turbinectomy for nasal discharge. A year prior to admission she developed drooping of the right upper lid, and right frontal headache. Trauma to the right frontal area had occurred from bumping her forehead repeatedly against the frame of the upper bunk of a double deck bed.

Examination showed ptosis of right upper lid and marked limitation of upward movement of the right eye. Some crusting at the site of the turbinectomy was the only nasal finding. Systemic examination revealed diabetes, hypertension and secondary anemia.

Roentgen examination showed a large area of decreased density adjacent to the upper border of the right orbit near the outer canthus measuring 3 cm. in diameter, which was circular and smooth. The frontal sinuses appeared clouded and were not definitely outlined until an iodized oil study was done. This opaque study revealed irregular filling of

the frontal sinus due to hyperplastic mucosa, and showed it separated from the round radiolucent area (see Fig. 5).

External opening of the sinus revealed a large lobulated cavity lined by a thickened adherent membrane which was separated from the round cystic area by a bony wall. The supraorbital plate was absent in its lateral half, where a greenish-gray cystic mass encroached upon the orbital space. The cystic cavity was without any epithelial lining and con-

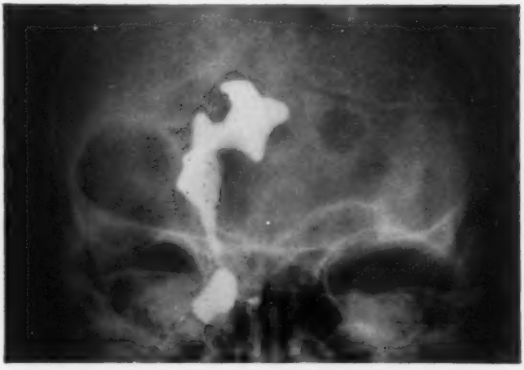

Fig. 5. Roentgenogram of opaque study showing irregular filling of the frontal sinus due to hyperplastic mucosa and its separation from the round radiolucent area which was the site of the traumatic cyst.

tained granulation tissue intermingled with a glue-like substance. Culture of pus from the frontal sinus showed staph-aureus and non-hemolytic streptococcus, but culture of the material from the cyst showed no growth. This was a dual lesion, a frontal bone cyst resulting from trauma, as described by Geschickter and Copeland,[1] and chronic frontal sinus disease.

Frontal sinus disease manifesting characteristic clinical symptoms and rhinoscopic findings was observed in only six patients, and they all complained of nasal discharge, nasal obstruction and headache or localized pain and tenderness over the frontal sinus. All of them had suppurative infection of the sinus, and all showed characteristic Roentgen findings and clinical manifestations of the disease. The small number

of such patients is worthy of comment, because it indicates how greatly chemotherapy and antibiotics have controlled suppurative infection in these sinuses.

My observations on this group are limited to brief comments on two patients: the first, a man age 57, with a history of chronic sinus infection extending over many years. He was subject to acute attacks characterized by frontal headache, purulent nasal discharge and nasal obstruction which were usually relieved with antibotic therapy and local nasal treatment; however, during one attack he developed edema and ptosis of the upper lid, edema of the lower lid, injection of the bulb, lachrymation and marked tenderness at the mid-point of the right brow, in addition to the nasal symptoms. These symptoms persisted and became worse, in spite of antibiotic therapy for a period of two weeks.

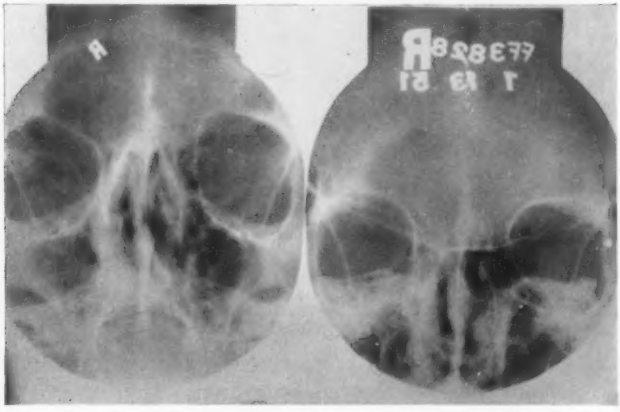

Fig. 6. Roentgenogram showing increased density of right antrum, right ethmoid and both frontal sinuses and in addition the floor of the right frontal thinned out and depressed toward the orbit from an expanding fluid mass within the sinus—a finding characteristic of mucocele.

The Roentgen film showed increased density of the right antrum, right ethmoid and both frontal sinuses. In addition, the bony elements of the floor of the right frontal appeared thinned out and depressed toward the orbit from an expanding fluid mass within the sinus, a finding characteristic of the mucocele-like (see Fig. 6) lesion observed in the first six patients.

External operation revealed a large erosion or perforation of the sinus floor with its muco-periostal lining bulging into the orbit, and when this was incised the cavity of the sinus was found filled with thick cream-like pus. A small probe was passed through the naso-frontal duct from the nasal cavity, showing it to be patulous but partially obstructed by swollen hyperplastic membrane. This obstruction was sufficient to put the pus under pressure, and cause expansion and erosion of the sinus floor, and his cure depended upon its removal regardless of antibiotic therapy.

The second patient, a 22-year-old man with acute fulminating frontal sinus infection, the result of swimming, complicated by staphylococcus bacteremia, was of interest because he illustrated the miraculous results that are possible with chemotherapy and biotherapy supplemented by a simple surgical procedure to promote drainage, in contrast to the futility of all therapeutic measures before the era of chemotherapy and antibiotics. This infection began as an ordinary head cold following swimming, but within 24 hours sudden severe pain occurred in and around the left eye, and he felt very ill. The symptoms increased rapidly, and within 48 hours he had a temperature of 104°, was very toxic, and had severe pain in his right hip. He was admitted to the hospital, and examination revealed acute suppurative infection of the left frontal sinus with pus draining from the naso-frontal duct.

In addition, he had a metastatic infection of the right hip. Roentgen examination confirmed the frontal sinus infection, and blood culture revealed a very heavy growth of staphylococcus aureus, hemolyticus, which was resistant to penicillin, terramycin and streptomycin, but was sensitive to aureomycin. Treatment consisted of aureomycin 500 mg. sixth hour, erythromycin 300 mg. sixth hour, and gantrisin 1 gm. fourth hour. In addition, nasal drainage was supplemented by removing a portion of the sinus floor through a small external incision and inserting a polyethylene tube, through which the sinus was irrigated with normal saline. The sinus infection cleared up within a week, but it required 37 days of chemo-

therapy and biotherapy to clear the blood stream, during which he took a total of 23,900 mg. of erythromycin, 16,500 mg. aureomycin and 150 mg. gantrisin.

The role of surgery was secondary to that of the chemotherapy and biotherapy in this patient, but I believe it played an important part because it assured free drainage and prevented expanding pressure from the accumulation of a purulent mass within the sinus cavity.

SUMMARY.

These observations indicate that the majority of patients with frontal sinus disease, due to a wide variety of lesions, are free of nasal symptoms and signs; in fact, eye symptoms and ocular findings were much more important in diagnosis than nasal symptoms and rhinoscopic findings. Delay in making an accurate diagnosis and adopting adequate therapy was a notable feature of the management of many of these patients, and they were subjected to futile eye surgery, useless Roentgen therapy, ineffectual biotherapy and chemotherapy, and also incision of overlying soft tissue abscesses which merely served to create discharging fistulas and scar tissue deformities before the true nature of the disease was recognized. Such delay indicates that nasal symptoms are considered by patient and practitioner to be the cardinal evidence of sinus diseases, and when they are absent investigation and treatment are apt to be directed to other fields than the sinuses.

These observations also illustrate that chemotherapy and the antibiotics have altered the course of suppurative infection in the frontal sinus, so that external surgery is required chiefly as a supplementary measure in its treatment; however, there is a primary need for surgery when mechanical obstruction of the sinus outlet is the basis for the pathology

and clinical manifestations of the disease, and antibiotics will fail to prevent or cure it without relieving this obstruction. Consequently these two important therapeutic agents should not be considered in any way as rivals, but as being complementary to each other. It is not a question of giving one and then if it fails resorting to the other; instead, adequate therapy requires skill in the use of both.

REFERENCES.

1. GESCHICKTER, CHARLES F., and COPELAND, MURRAY: Tumors of Bone. 3rd edition, p. 64, 1949. J. B. Lippincott & Co., Philadelphia, Pa.

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CORRECTION.

In the November, 1953, issue (Vol. LXIII, No. 11, pp. 1089-1095), page 1092, line 12, the paper by Dr. Felix E. LeBorgne of Ibicuy 1210, Montevideo, Uruguay, reads "originating in the free border." This should read "originating above the free border . . . . ".

BACTERIOLOGY AND VIROLOGY OF ACUTE INFECTIONS OF THE RESPIRATORY TRACT.*†

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(By Invitation).
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Much has been said and written about various microbial agents and their relationships to acute respiratory infections. It would seem worthwhile to depart from this rather stereotyped approach occasionally and to weigh certain fundamentals with broad applications. Such is the object of this treatise, and, accordingly, a minimum of space will be devoted to a detailed discussion of any particular genus or species. Conversely fundamental characteristics encompassing many microbial agents will be given proportionately larger consideration.

Microbes exist as populations. This statement is significant whether one deals with a mixed normal flora or an alleged pure culture of infection. While the normal flora of the nose, throat and mouth varies some from one individual to another, the fact remains that it is a mixture of not only different species but also of different genera. The ratio of one species or genus to another may be important. Several instances seem to illustrate the case in point. *Hemophilus influenzae* was considered to be a relatively uncommon component of the normal oral cavity before the use of penicillin allowed it to be cultured in high incidence.[1] *Candida albicans* is found in varying numbers quite frequently in apparently normal mouths.[2] The increase in candida infections may be related to an upset in the balance of the microbial flora although there are other possible explanations.[3] Acute toxic deaths following prolonged antibiotic therapy with penicillin and streptomycin in which staphylococci were present in large numbers

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* Read at the meeting of the Middle Section of the American Laryngological, Rhinological, and Otological Society, Inc., St. Louis, Mo., January 18, 1954.

† From the Department of Bacteriology, The State University of Iowa.

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have been reported.[4] Here again one might suggest that an upset in the total population may have played a part.

While antibiotics have been used to illustrate the promotion of disparity among the total microbial population, other factors may also play a part: the influence of diet, pH, natural microbial antagonism and the immune state may also influence normal flora.

Most studies of microbial populations have concerned bacteria; nevertheless, some contributions relative to viruses lead us to believe that interrelations between viruses and bacteria and between viruses and viruses also exist. Two different types of influenza viruses have been demonstrated to propagate side by side through numerous serial passages in the chick embryo.[5] Competition, among homologous and heterologous viruses, for perhaps the same host cell receptor, has been suggested. That certain viruses can block or partially inhibit the propagation of others in the same locale has been repeatedly demonstrated.[6]

In addition to alterations in total populations a single species is beset with much potential variation. In a very restricted sense one could question whether a pure culture ever exists. In a practical sense it does; however, the inherent adaptability and proneness to mutate or vary among microbes makes one acknowledge that he deals with a very labile progeny, even if they spring from a single cell isolation.

While the incidence of labile cells among the total population may be low, these cells are none the less important. These are the cells that so readily adapt themselves to therapy or other would-be adverse situations. The fact that such individuals may exist in low ratios such as 1:10,000 or greater loses its significance when microbial growth rates are considered. Bacteria such as *Bacillus megatherium* and *Escherichia coli* have been known to reproduce themselves in 9 and 16.5 minutes, respectively.[7,8] Since this multiplication is geometric the uncommon cell today, that is the 1:10,000, literally becomes the common cell tomorrow. Any of the repressive influences listed above may promote this very change.

Many changes among bacteria are dynamic and not at all permanent. The ebb and flow of these changing circumstances suggest an equilibrium propensity on the part of the microbe. Some observers have noted this change in the course of antibiotic therapy. In such instances staphylococci isolated from various infectious processes have increased in resistance from an early 10 per cent resistance to a 60 per cent resistant incidence plateau. When the antibiotic in questions was withdrawn from the therapeutic armamentarium the resistant incidence fell to approximately 40 per cent but returned to 60 per cent again when penicillin was reinstated.[9] The response of bacteria to antibiotics undoubtedly is not so simple as the two extremes noted above. Already a third state has been described among the Neisseriaceae resulting in streptomycin dependent as well as resistant and susceptible strains.[10] Perhaps the more accurate concept would involve a spectrum of resistance or/and susceptibility.

An even more complicated change than just antibiotic resistance has been shown experimentally. *Diplococcus pneumoniae* has been altered antigenically and in resistance. Under the influence of the appropriate desoxyribonucleotidase and antibiotic the organism was changed to a new type by virtue of synthesizing a new capsule and a new state of resistance by changing from a susceptible to a resistant organism.[11] If such broad biological adaptation could occur *in vivo* the significance becomes apparent. This would offer the microbe two avenues of escape.

While viruses present a considerably more difficult group with which to work, some of their potentialities in regard to genetic recombinations have been studied. Viruses of different characteristics propagated simultaneously in the same medium seem to produce progeny possessing characteristics of both original strains.[12] This if it occurs under natural conditions, could be a factor in explaining some of the vagrancies involved in immunizing against influenza. This would not have to be the only influence involved, however. Investigators interested in viruses attacking bacteria have demonstrated a number of factors influencing bacterial virus propagation and have revealed a number of instances of genetice recombinations.[13]

The influence of antibiotics on some of the larger viruses with regard to their growth cycle has provided interesting material for speculation. Members of the Psittacosis-Lymphogranuloma venereum group appear to go through a definite growth cycle.[14] Large initial bodies appear within the cytoplasm of infected cells. These in turn break down to progressively smaller units, the elementary bodies. With cell rupture the elementary bodies are released to parasitize new cells. It is interesting to note that penicillin appears to exert its action early in the cycle, and broad spectrum antibiotics exert their effects later in the cycle.[15] Medium size and small viruses may also have cycles of growth, though this is sometimes difficult to establish. Influenza virus, a medium size virus of about 100 mμ., apparently goes through progressive changes as it is propagated. In the early stages during multiplication it is difficult to demonstrate, so much so that it could be interpreted to exist in a "masked form.[16] Subsequently it becomes more complex, finally to be released from the host cell in the form about which reference is usually made.

The cycle or progressive change in the virus during its propagation does have other than academic interest. The virus may vary in antigenicity, infectiousness and toxicity, and perhaps in other propensities during these various stages. One could readily consider the possibility that this in turn may influence the results of therapy, artificial active immunization and carrier stages. In the light of the work on the psittacosis virus one might consider that other viruses may have a vulnerable point of attack for some growth-inhibiting agent.

If, at a given point in the reproductive cycle, the virus were antigenically satisfactory, that is to say, capable of stimulating a successful immune state and yet lacking in toxic properties, this would constitute an advance in immunization technique. One could raise the question as to which portion of the cycle might most successfully be immunized against. Does the antigenic variation phenomenon stem from some particular part of the cycle? These questions not only justify the research aimed at understanding the method or methods of

viral propagation but may in addition yield information of very practical significance.

The masked or immature forms of viruses, if they exist generally, may provide some explanation of the whereabouts of certain viruses in interepidemic periods. Few would question the existence and importance of human carriers of bacterial pathogens. Proving the existence of human carriers of viral pathogens presents a more difficult problem. A number of pathogenic bacteria are known to grow in their specific antiserum. In the presence of specific antiserum the virus seems to be at a disadvantage propagation-wise. A neutralized virus may fix itself to a host cell, but its subsequent multiplication, if adequately neutralized, may well be equivocal. The problem of readily demonstrating human carriers of pathogenic viruses, therefore, becomes a real one.

Recurrent herpes not uncommonly makes its appearance in the same area of the skin or mucocutaneous border. This fact has led many to question whether or not the virus might not be existing in several parasitized cells perhaps protected in this fashion from the effects of body defense, existing, as it were, in a balanced state with the host, wherein the parasite propagates insufficiently to cause clinical symptoms, yet is not completely eradicated from the host. This thesis receives considerable support from a chance observation in which latent herpes encephalitis appeared to be provoked following an hypersensitive reaction. In this instance rabbits receiving insufficient virus to produce clinical encephalitis were used six months subsequently to demonstrate anaphylaxis. Seven days following the anaphylactic experiments the rabbits came down with fatal herpes encephalitis. In this same regard the potentiating effect of histamine was experimentally demonstrated.[17]

Examples of this kind, though not numerous, are of sufficient occurrence to focus attention on the possibility of hypersensitive reactions serving as a percussion cap to explode some infectious diseases. The most likely area of applicability would be among those diseases given to recurrence and perhaps also those in which subclinical infections occurred, establishing a quasi-immune state.

The very interesting situation prevailing in swine influenza leads one to speculate about this same situation. The influenza virus in its masked form appears to reside in the lung worm larva which in turn resides in the earthworm. When swine eat earthworms the lung worm larvae find their way to the lungs of the swine. Unless the swine be provoked by some special method no infection results. In nature this may be a meteorological provocation since swine influenza usually becomes apparent during the first cold spell of the season; nevertheless, the situation has been precipitated following repeated intramuscular inoculations of bacteria.[18] The possibility that an induced hypersensitivity may have played a part in unmasking the viral agent must be considered.

A point of importance to be established is whether an infectious agent such as a virus is necessarily destroyed in the immune individual. Experimental investigation tends to back up the idea that virus may remain alive in the convalescent immune animal for some time. Table I summarizing the results of such an experiment appears below.

TABLE I.—THE RECOVERY OF INFLUENZAL VIRUS, TYPE APR8 FROM CONVALESCENT MICE WITH AND WITHOUT THE USE OF CONCENTRATED, INACTIVE, HOMOLOGOUS VIRUS.

	DAYS AFTER INOCULATION									
	5	6	7	8	9	10	11	12	13	14
Using concentrated inactive virus	+	+	+	+	+	—	—	+	*	+
Using saline	+	+	+	+	+	—	—	—		—

* Omitted to extend the experiment another day.

The concentrated heat inactivated homologous virus is believed to cause a redistribution of the neutralizing antibody. By tying up a large amount of the available antibody it is suggested some of the antibody neutralizing the active virus is dissociated, thus allowing the previously neutralized active virus to become infective. This same situation is believed to be operative even in the hyperimmune animal. Experimentally it is possible to show viable virus present for at least two days longer by the reactivation technique than without it.[19] The above findings suggest interesting possibilities. If the virus is capable of surviving in the convalescent immune or hyperimmune individual for a sufficient length of time, it would be faced with less overwhelming odds as far as anti-

body is concerned. If the antibody titer dropped low enough, a recurrence of the infection might occur. The reoccurrence of the infection should stimulate a secondary immune response allowing the exaccerbation to be short lived. In this vein of thought one cannot help but see the possible fulfillment in such infections as primary and recurrent herpes. The severity of the primary attack is well known as is the relatively milder episodes in the recurrent disease.

To return to the proposition of convalescent carriers of viral diseases, what situation might prevail here? If the immune carrier discharged neutralized virus to susceptible recipients, would the neutralized virus "take root and grow?" Since they were not capable of inciting clinically obvious disease in the carrier would one expect them to in the recipient? To speculate in this area one cannot afford to overlook the facts concerning virus neutralization. A virus may be underneutralized, just neutralized, or overneutralized with its antibody. An underneutralized virus is infectious, a just neutralized virus is not; but the balance is precarious. The investigator need but to dilute the just neutral mixture and it becomes infective.[20] Enzymatic destruction of a small portion of the antibody or dissociation of the antibody in the presence of a small amount of active, inactive, or underneutralized homologous antigen may upset this balance.[21,22] While antigen-antibody reactions are specific, the fact that the same antigen may be found in widely separated biological situations must not be overlooked.

If a just neutral virus lands in the nonimmune recipient the factor of dilution should shortly become effective, for the gradient here would encourage dissociation, not to mention dilution with body secretions.

There is another fact concerning the interaction of the virus and its specific antibody which is well known but not commonly applied to immunological philosophy. The antibody function involved in the complement-fixation tests is not always the same as that involved in the immune state. This is true also in some other *in vitro* antibody tests; however, *in vitro* antibody assays are usually more convenient than those involving the direct challenge of the experimental an-

imal, and, therefore, they are more commonly employed in immunological studies. The presence of complement-fixing antibodies in the individual's serum usually indicates that he has encountered the infectious agent in question and, therefore, should be better able to deal with it than the unexposed; however, at the moment of exposure, he would not necessarily have sufficient protective antibody titer to prevent onset of the infection. Hence, immunological results, like any other results concerned with the practice of medicine, should be subject to careful examination prior to interpretation.

Arbitrarily and usually for convenience, the hypersensitive and immune states are separated. While this may be justified for the purpose of discussion one could question its general advisability. A case in point is the readiness with which immunity function may be overlooked in antitoxin prophylaxis. When the physician administers horse tetanus or diphtheria antitoxins his concern is usually two-fold; first, he is concerned with rendering the patient passively immune; second, he is concerned with the possibility of his patient's being hypersensitive to horse serum. It is well known that to administer horse serum to the individual hypersensitive to horse protein may result in anaphylactic shock. Even if this is his first exposure, serum sickness may present a problem. The third concern is often overlooked. The individual who has once received horse serum is in a sense immune to it, in that he is capable of eliminating it at a faster rate than someone who has not previously been exposed to this very good foreign protein antigen.[23] This fact becomes of practical significance under any circumstances where additional doses of prophylactic serum seems advisable. Generally speaking, one should think of the hypersensitive and immune states concomitantly lest he stress one aspect to the exclusion of the other.

Finally, close cooperation between physician and microbiologist is not only desirable but also necessary for best results. Such an ideal situation goes far in deciding the significance of laboratory findings. To illustrate this a not uncommon problem may be examined. The diagnosis of pulmonary mycosis usually requires considerable study to substantiate it

definitely. The demonstration of sulphur granules in the patient's sputum might lead one to a hasty diagnosis of actinomycosis. The sources of these granules, however, may be chronically infected teeth or tonsils. Repeated rather than an occasional demonstration of the granules and demonstrating them in a specimen obtained by a bronchoscope adds weight to the findings. The exclusion of other possible pathogens such as *Mycobacterium tuberculosis* lends additional weight to the diagnosis. The fact is that some potential pathogens may exist as opportunists among saprophytic normal flora, and the relative incidence of any one organism in a given area may be upset following antibiotic therapy; therefore, laboratory findings should always be weighed carefully in each instance so as to make the most intelligent use of them.

It is difficult to summarize fundamentals for they in themselves are essentially not reducible; however, the philosophical attitude toward the fundamentals referred to above perhaps can be epitomized. Any physician dealing with infections does well to keep in mind at all times that he deals with one of the most versatile antagonists of mankind. Any physician dealing with hypersensitive states must raise his eyes to the horizon of immunology realizing that it is a broad and interrelated field. Last but not least, close cooperation between the basic scientist and practicing physician is mutually beneficial.

BIBLIOGRAPHY.

1. Fleming, A.: On the Antibacterial Action of Cultures of a Penicillium with Special Referrence to Their Use in the Isolation of *B. Influenzae*. *Brit. Jour. Exper. Path.*, 10:226, 1929.

2. Todd, R. L.: Studies on Yeast-like Organisms Isolated from the Mouths and Throats of Normal Persons. *Amer. Jour. Hyg.*, 25:212, 1937.

3. Woods, J. W.; Manning, I. H., Jr., and Patterson, C. N.: Monilial Infections Complicating the Therapeutic Use of Antibiotics. *J.A.M.A.*, 145:207, 1951.

4. Jackson, G. G., et al.: Terramycin Therapy of Pneumonia: Clinical and Bacteriologic Studies in 91 Cases. *Ann. Int. Med.*, 35:1175, 1951.

5. Sugg, J. Y.: Further Studies on Serial Passage of a Mixture of Influenza Viruses in Embryonated Eggs. *Proc. Soc. Exper. Biol. and Med.*, 76:199, 1951.

6. Henle, W.: Interference Phenomena Between Animal Viruses: A Review. *Jour. Immunol.*, 64:203, 1950.

7. Henrici, A. T., and Ordal, E. J.: The Biology of Bacteria. P. 169, 3rd Edition, D. C. Heath and Co., 1948.

8. Porter, J. R.: Bacterial Chemistry and Physiology, P. 129, John Wiley and Sons, Inc., 1946.

9. Needham, G. M., and Nichols, D. R.: Recent Changes in Sensitivity of Micrococcus Pyogenes to Various Antibiotic Agents. *Jour. Lab. and Clin. Med.*, 41:150, 1953.

10. Miller, C. P., and Bohnhoff, M.: Two Streptomycin Variants of Meningococcus. *Jour. Bact.*, 54:467, 1947.

11. Hotchkiss, R. D.: Transfer of Penicillin Resistance in Pneumococci by the Desoxyribonucleate Derived from Resistant Cultures. *Cold Spring Harbor Symposia on Quantitative Biology*, 15:457, 1951.

12. Hershey, A. D., and Chase, M.: Genetic Recombination and Heterozygosis in Bacteriophage. *Cold Spring Harbor Symposium on Quantitative Biology*, 16, 1951.

13. Hershey, A. D., and Davidson, H.: Allelic and Nonallelic Genes Controlling Host Specificity in a Bacteriophage. *Genetics*, 36:667, 1951.

14. Bedson, S. P., and Bland, J. O. W.: Developmental Forms of Psittacosis Virus. *Brit. Jour. Exper. Path.*, 15:243, 1934.

15. Gogolak, F. M., and Weiss, E.: The Effect of Antibiotics on Agents of the Psittacosis-Lymphogranuloma Group. II. The Effect of Aureomycin. *Jour. Infect. Dis.*, 87:264, 1950.

16. Henle, W., and Henle, G.: Studies on Host Virus Interaction in the Chick Embryo-Influenza Virus System. III. Developments of Infectivity Hemagglutination and Complement-Fixation Activities During the First Infectious Cycle. *Jour. Exper. Med.*, 90:23, 1949.

17. Good, R. A., and Campbell, B.: Potentiating Effect of Anaphylaxis and Histamine Shock Upon Herpes Simplex Infection in Rabbits. *Proc. Soc. Exper. Biol. and Med.*, 59:305, 1945.

18. Shope, R. E.: The Swine Lungworm as a Reservoir and Intermediate Host for Swine Influenza Virus. III. Factors Influencing Transmission of the Virus and the Provocation of Influenza. *Jour. Exp. Med.*, 77:111, 1943.

19. McKee, A. P., and Hale, W. M.: Reactivation of Overneutralized Influenza Virus in Chicken Embryos. *Jour. Immunol.*, 58:141, 1948.

20. Andrewes, C. H.: The Action of Immune Serum on Vaccinia and Virus III, *in Vitro*. *Jour. Path. and Bact.*, 31:671, 1928.

21. Kalmanson, G. M., and Bronfenbrenner, J.: Restoration of Activity of Neutralized Biologic Agents by Removal of the Antibody with Papain. *Jour. Immunol.*, 47:287, 1943.

22. McKee, A. P., and Hale, W. M.: Reactivation of Overneutralized Mixtures of Influenza Virus and Antibody. *Jour. Immunol.*, 54:233, 243, 1946.

23. Glenny, A. T., and Hopkins, B. E.: Duration of Passive Immunity. *Jour. Hyg. Camb.*, 21:142, 1922.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

PROGRAM.

Tuesday, May 25, 1954

Time

9:00 A.M. *Business Meeting:*
Election of Candidates.
Election of Nominating Committee.
Induction of Newly Elected Fellows.

9:30 A.M. *Presidential Remarks:*
LeRoy A. Schall, M.D., Boston, Mass.

9:40 A.M. *Introduction of the Guest of Honor:*
J. Parsons Schaeffer, M.D., Professor Emeritus of Anatomy, Jefferson Medical College, Philadelphia, Pa.

SCIENTIFIC SESSION.

9:50 A.M. *1.* Mortalities and Morbidities Following 20,000 Tonsil and Adenoidectomies: George O. Cummings, C.D., Portland, Maine.

10:10 A.M. Open Discussion.

10:20 A.M. *2.* Meniere's Disease—An Unrehearsed Table Discussion (No speeches):
Julius Lempert, M.D., New York, N. Y., (Moderator).
Mr. Terence Cawthorne, London, England.
Kenneth M. Day, M.D., Pittsburgh, Pa.
John R. Lindsay, M.D., Chicago, Ill.
Oral or written questions may be submitted from the floor.

11:00 A.M. *3.* The Study of Ciliated Epithelium with the Electron Microscope: (By Invitation).
Don W. Fawcett, M.D., Boston, Mass., (By Invitation)

11:30 A.M. Open Discussion.

11:40 A.M. 15-MINUTE RECESS.

11:55 A.M. *4.* Progressive Oral Gangrene Due to Acatalasemia—Colored Motion Picture:
Shigeo Takahara, M.D., Okayama University Medical School, Japan (By Invitation).

Discussion:
William Dameshake, M.D., Boston, Mass., (By Invitation).

12:25 P.M. *5.* The Role of Triangulation Roentgenoscopy as a Method of Guidance in the Removal of Opaque Foreign Bodies Beyond Bronchoscopic Vision:
Alfred A. Dorenbusch, M.D., Charlotte, N. C., (Candidate's Thesis).
No Discussion.

Wednesday, May 26, 1954

Time

9:00 A.M. *"In Memoriam."*

Business Meeting:

Report of the Nominating Committee.

New and Miscellaneous Business.

SCIENTIFIC SESSION.

9:30 A.M. *6.* Leverage System of Suspension-Fixation Laryngoscopy (with Motion Picture):
Robert B. Lewy, M.D., Chicago, Ill.

9:50 A.M. Open Discussion.

10:00 A.M. *7.* Industrial Noise and Occupational Deafness:
Mr. Harry Nelson, Director of Compensation, Industrial Commission State of Wisconsin, Madison, Wis., (By Invitation).

10:45 A.M. Question Period.

11:00 A.M. *8.* The Surgical Treatment of the Atresia Auris Congenita. A Clinical and Histological Report: Professor Luzius Ruedi, Zurich, Switzerland.

11:30 A.M. Discussion:
Howard P. House, M.D., Los Angeles, Calif.

11:40 A.M. 15-MINUTE RECESS.

11:55 A.M. *9.* Headache Mechanisms:
Harold G. Wolff, M.D., New York, N. Y., (By Invitation).
No Discussion.

Thursday, May 27, 1954

Time

9:00 A.M. *Business Meeting:*
Election of Council.
Five-Minute Council Meeting.
Presentation of New Instruments.

SCIENTIFIC SESSION.

9:30 A.M. *10.* An Experimental Study of the Small Blood Vessel of the Spiral Ligament and Stria Vascularis of Living Guinea Pigs During Anaphylaxis:
Francis L. Weille, M.D., Boston, Mass.

9:50 A.M. Open Discussion.

10:00 A.M. *11.* A Half Century of Triological Transactions:
Lyman G. Richards, M.D., Wellesley Hills, Mass.
No Discussion.

10:30 A.M. *12.* The Emotional Preparation of Children for Surgery: Television Kinescope:
No Discussion.

11:00 A.M. 15-MINUTE RECESS.

11:15 A.M. *13.* Neoplasms of the Middle Ear and Mastoid:
Wesley H. Bradley, M.D., Syracuse, N. Y., (By Invitation).
James H. Maxwell, M.D., Ann Arbor, Mich.

11:35 A.M. Discussion:
Daniel Miller, M.D., Boston, Mass.

11:45 A.M. *14.* Benign Malignant Lesions of the Larynx—A Motion Picture:
Gabriel Tucker, M.D., Philadelphia, Pa.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

April 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 106 So. 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model G-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Myte); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor, 2 tubes. Model A-330; 3 transistors.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 35, New York.

Audivox, Model 71; 3 transistors.
Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.
Manufacturer: Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32. Illinois.

Maico Transist-Ear, Model O; 3 transistors.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1, Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors.
Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarian Model F-22; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Radioear Model 820; 3 transistors.

Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-25; 3 transistors.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn. Distributor: Sears, Roebuck & Co., 925 South Homan Ave., Chicago 7, Illinois.

Sonotone Model 1010; 1 transistor, 2 tubes.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Me.
Vice-President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Boston, Mass., May 23-24, 1954.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Gordon F. Harkness, Davenport, Iowa.
First Vice-President: Claude C. Cody, Houston, Tex.
Second Vice-President: Daniel S. Cunning, New York, N. Y.
Secretary: Harry P. Schenck, Philadelphia, Pa.
Treasurer: Fred W. Dixon, Cleveland, Ohio.
Meeting: Statler Hotel, Boston, Mass., afternoon, May 27; all day, May 28.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston, Mass.
President-Elect: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Boston, Mass., May 25-27, 1954. (Mornings only.)

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Dean Lierle, Iowa City, Iowa.
Vice-Chairman: Dr. Fred W. Dixon, Rose Bldg., Cleveland, Ohio.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.
Meeting: San Francisco, Calif., June 21-25, 1954.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago 11, Ill.
President-Elect: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Waldorf-Astoria, New York City, Sept. 19-24, 1954.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Statler Hotel, Boston, Mass., May 17-22, 1954.
Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Edwin N. Broyles, 1100 No. Charles St., Baltimore 1, Md.
Secretary: Dr. F. Johnson Putney, 255 So. 17th St., Philadelphia (3) Pa.
Meeting: Statler Hotel, Boston, Mass. (Afternoons) May 25-26, 1954.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Theo. E. Walsh, 640 So. Kingshighway, St. Louis 10, Mo.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Albert D. Ruedemann, 1633 David Whitney Bldg., Detroit 26, Mich.
President-Elect: Dr. F. Lambert McGannon, 14900 Detroit Ave., Lakewood 9, Ohio.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Waldorf-Astoria, New York City, September, 1954.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Norris J. Heckel, Chicago, Ill.
President-Elect: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting: Chicago, Ill., Sept. 22-24, 1954.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Peter N Pastore, Richmond, Va.
President-Elect: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
Vice-President: Dr. H. L. Mitchell, Lexington, Va.
Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Harold Owens, M.D.
Secretary-Treasurer: Robert A. Norene, M.D.
Chairman of Section on Ophthalmology: Sol Rome, M.D.
Secretary of Section on Ophthalmology: Wendell C. Irvine, M.D.
Chairman of Section on Otolaryngology: Max E. Pohlman, M.D.
Secretary of Section on Otolaryngology: Herschel H. Burston, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Nievert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Fleit, 66 Park Ave., New York (16), N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Cecil Swann, Asheville, N. C.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David S. Asbill, Columbia, S. Car.
Vice-President: Dr. John McLean, Greenville, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.
Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Harry P. Schenck.
Vice-President: Dr. William J. Hitschler.
Treasurer: Dr. Chevalier L. Jackson.
Secretary: Dr. John J. O'Keefe.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. M. Valentine Miller, Dr. Charles E. Towson, Dr. Thomas F. Furlong, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. Robert Black, 401 Medical Arts Bldg., Winnipeg, Manitoba.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Harrison Hot Springs Spa, Harrison Hot Springs, B. C.
Time: June 13-15, 1954.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Oscar Marchman, Jr., Dallas, Texas.
Secretary-Treasurer: Dr. Morris F. Waldman, Dallas, Texas.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretario del Exterior: Dr. Juan Manuel Tato.
Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Díaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

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FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: U. S. A., 1957.

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